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## MOTOR DISORDERS OF THE CENTRAL NERVOUS SYSTEM AND THEIR SIGNIFICANCE FOR SPEECH.*†

### PART I. — CEREBRAL AND CEREBELLAR DYSARTHRIAS.

DR. PAUL J. ZENTAY, St. Louis.

The purpose of this paper is to discuss the manifold motor disturbances of speech that occur with lesions of the various motor centres and tracts in the central nervous system. These disturbances may conveniently be grouped together under the name of dysarthrias of speech. Their analysis and clear understanding in the light of newer knowledge of the pathophysiology of the central nervous system are extremely important for all who are interested in the study and correction of disorders of speech. Insight gained into these problems must contribute greatly to the evaluation of the potentialities and limitations of speech in a given case.

In the present discussion we shall ignore those disturbances on the highest functional levels that are known under the name of aphasia and apraxia. For practical purposes of this presentation we even have to presuppose that functions on these levels are more or less intact.

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*Read before the American Speech Correction Association, St. Louis, Dec. 30, 1936.

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Still, nothing appears more suitable as a starting point for our discussion than a quotation from Weisenburg's recent admirable work on aphasia: "Our views on localization, so far as it concerns language, differ from those largely held by the localizationists in that we believe that it is impossible to delimit areas for the language function or any of its processes. The statement of Hughlings Jackson, made almost 70 years ago, that 'To locate the damage which destroys speech and to locate speech are two different things,' is just as true today as it was then. It is evident that it is impossible to localize language. In the majority of individuals, language permeates the thought processes to such an extent that the one cannot be separated from the other, and for the present at least it is impossible to give an adequate explanation of intelligence, much less to localize it. That it is the result of the activity of the entire brain, however, there is no doubt."

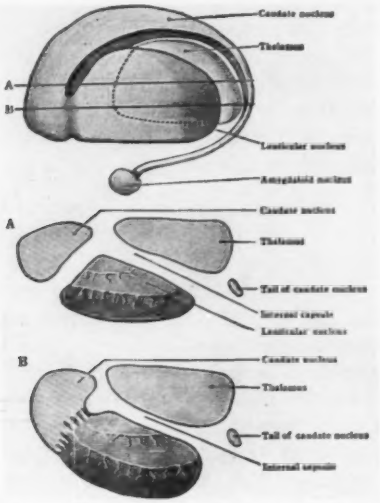
It is evident that in disorders on this level the most hopeful approach is the psychological. Below the cortical and subcortical level, on the other hand, speech function consists largely of the proper transmission and conduction of motor stimuli and, therefore, the most useful and practical method of study will be the neurophysiological approach. In such a study we necessarily have to bear in mind constantly that in speech as well as in other motor performances we are dealing with functions of the entire central nervous system. It also must be remembered that in any loss of motor function the final results will be dependent just as much on the lesion itself as on the parts intact and capable of compensation or substitution.

The motor impulses of speech originating in the so-called speech centres of the cortex are transmitted to the motor areas of the parietal and frontal lobes. From this point on they follow exactly the same pathways as any other conscious-voluntary motor innervation. These pathways are: *1.* Cortico-bulbar (pyramidal) tracts; *2.* cortico-strio-pallido-rubro-bulbar (extrapyramidal) tracts; *3.* fronto-ponto-cerebello-rubro-bulbar tracts.

As evident, all three of these tracts converge on the ultimate efferent lower motor neurons in the bulbar nuclei of the cranial nerves. It goes without saying that speech can be physiological only if the functioning of all three motor sys-

tems is intact. They not only have to function together harmoniously and simultaneously, but they must maintain mutually a proper tonus and balance. Lesions in any of the three systems lead to well defined disorders and will necessarily have also their concomitant psychological characteristics.

Due to the nature of the lesions usually affecting these motor systems, it is evident that speech disorders as a rule will form only part of a more widespread motor disturbance.




Diagrams of lateral view and sections of the nuclei of the corpus striatum with the internal capsule omitted. (A) and (B) below represent horizontal sections along the lines (A) and (B) in the figure above. The figure also shows the relative position of the thalamus and the thalamus and the amygdaloid nucleus (Jackson-Morris).

It is also easily understandable that speech disorders observed with such lesions will largely bear the same general characteristics as found with lesions in other parts of these motor systems. Lesions in any of the three motor systems may be present, of course, without any involvement of the tracts serving the functions of speech. On the other hand, the more extensive the lesions are, the more there is likelihood that speech also will be involved. Such an involvement of speech may present all possible degrees and all forms of individual variations. The descriptions in the further part of this presentation must be looked upon as generalizations and types, not as clinical entities.

*Cortico-bulbar Tract Lesions:* These are known also under the name of pyramidal tract lesion, upper motor neuron lesion, or spastic paralysis. They are due to a partial or complete interruption of the pathways at any point between the parietal lobe cortex and the nuclei of the cranial motor nerves. There are no separate pyramidal tracts known for speech. This function is served by the same identical cortical areas and tracts as other motor innervations going to the nuclei of the Vth, VIIth, IXth, XIth and XIIth cranial nerves. Any or all of these tracts may be involved in a pathological process and the resultant disturbance evidently will depend on the extent of the lesions. The closer the destructive process is to the nuclei, the more the picture will resemble that of pseudobulbar paralysis with a more or less complete loss of speech. The tracts to the nuclei of the IXth, XIth and XIIth nerve are particularly important in this respect.

If the pathological lesion is in the subcortical areas or. still more, if it is in the cortex itself, the anatomical conditions make it possible that at times only some of the above tracts are involved, or that the destruction is only partial. In such cases we are dealing with the various forms and degrees of the so-called pyramidal or spastic speech. The type of spastic speech observed is largely determined by the extent of the involvement of the muscles of the speech organs. Great differences will be noticeable if only the facial or the lingual or laryngeal muscles are affected or if all of them are paralyzed. The disturbance will be also decisively influenced by the presence of unilateral or bilateral lesions in the cortex or further below.

On the whole the spastic speech is characterized by the loss of conscious voluntary innervations and increase of muscle tonus and reflex irritability. It is a slow dragging speech with indistinct articulation, apparently requiring a great deal of effort and often accompanied by facial distortions. At times the slowness alternates with explosiveness.

Neurophysiology does not know of any possibility of compensation or substitution for functions lost in the conscious-voluntary pyramidal system; therefore, with such lesions the development of speech depends on the residues.

*Cortico-strio-pallido-rubro-bulbar Tracts (Extrapyramidal Innervation):* In analogy with other somatic motor functions the innervations to muscles of the speech organs travel

through these tracts simultaneously with those reaching the same muscles through the pyramidal tracts.

In the hierarchy of this system the striate body (caudate nucleus) occupies the highest position. It seems that the caudate nucleus and the subservient extrapyramidal system perform the following functions in the mechanism of speech: *a.* Regulating and properly distributing the tonus of muscles; *b.* coordinating and connecting with speech a host of ready and fully developed automatisms; *c.* regulating the necessary synkineses and essential coinnervations; *d.* controlling and regulating the pre-existing automatic motor functions, such as maintaining the required tone, rate and depth of respiration, pitch of voice, tempo of speech.

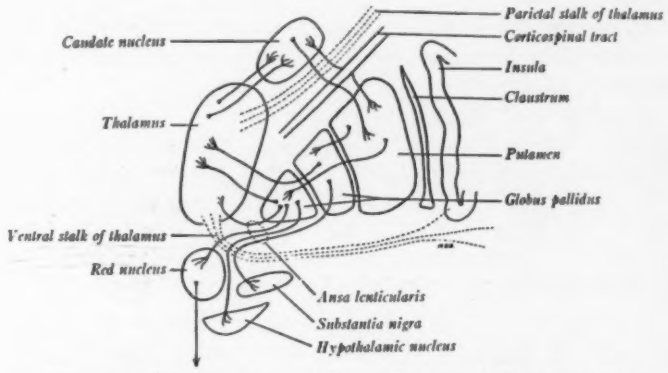



Diagram of the connections of the caudate and lenticular nuclei.

It is evident that all these innervations are vitally important for normal speech. They take place without any conscious effort and their significance becomes manifest when some kind of pathological process interferes with the functions of this system. The caudate nucleus apparently shows a definite somatotopic distribution of its functions. Lesions, therefore, will lead to various forms of disturbances dependent on their localization. The same is true of lesions affecting other parts of the extrapyradimal system. The common characteristics of these speech disturbances will be that they always appear in the form of dysarthrias and that they usually are associated with other manifestations of so-called extrapyramidal syndromes.

Two large groups of such syndromes are known: *a.* Hypotonic-hyperkinetic forms, such as athetosis, chorea, torsion spasm; *b.* hypertonic-hypokinetic forms, such as the Parkinsonian syndrome. In both of these groups the speech shows very definite characteristics. In the first group we find hyperkinesis of the vocal cords, involuntary movements of the diaphragm, and incoordination of various movements. The result of which is that the voice will be weak and pitch will suffer, leading in extreme cases to a complete disability of speech.

In the second group there is an extreme slowing down of movement, due to difficulty of "breaking through" of innervations. Speech in such cases is hesitant, halting, often interrupted or delayed for many seconds or minutes; it is also lacking in modulation and in advanced cases becomes more and more blurred.

It seems from all the above that the striopallidum occupies an extremely important position in the pathophysiology of speech.

Liepman and Raecke in their hypothesis postulated the existence of some kind of a mechanism interpolated between kinesthetic memory or mnesis and the effective motor apparatus of speech. Lesions of such a mechanism should—according to this theory — lead to dysarthrias of speech. Such an interpolated mechanism is represented unquestionably at the present state of our knowledge by the striate body.

Paul Schilder developed an interesting hypothesis, basing the same on facts gathered from the study of dysarthrias of speech in extrapyramidal lesions. He discovers many points of relations and similarities between disorders resulting from lesions in the striopallidum or in the mesencephalon and between the problem of stuttering. "Here we may find an important lead for the explanation of the pathophysiology of stuttering. Without having any reason to doubt the psychogenic origin of stuttering, it must be linked to functions of certain parts of the brain. Careful psychologic studies of organic brain lesions will necessarily bring about the desire to explain the neuroses on the basis of brain pathology.

"In this sense we may classify stuttering together with tics as 'striate body neurosis'."

*Fronto-ponto-cerebellar Tracts:* The principal functions of this system are regulating muscle tonus and balance. coordination, controlling the proper measure of motions.

Without doubt a complicated working together of the various muscle groups and apparatus in speech is absolutely necessary. Still the concepts gained from the coordination of body musculature are very inadequate.

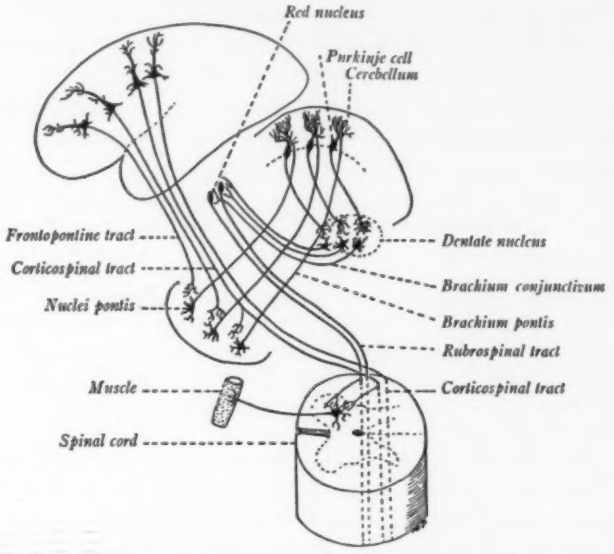



Fig. 242. The cortico-ponto-cerebellar and cerebello-rubro-spinal paths (modified from Cajal).

Dysarthrias of speech in cerebellar lesions may be described under the following headings:

*a. Ataxic Speech:* It is due to lesions of the coordinating cerebellar tracts for articulation, respiration and phonation. These tracts run through the subthalmic region to the motor centres of phonation localized in the medulla.

Motor incoordination may involve various parts of the complex mechanism of speech in varying degree; *i. e.*, articulation, respiration and phonation may be interfered with in an uneven measure.

*b. Adiadochokinesis:* Here the attained contraction of muscles decreases very rapidly. On the extremities this manifests itself so that repeated movements gradually decrease in intensity and finally cease. In speech similar manifestations

may be noted. In spontaneous speaking adiadochokinesis may be overcompensated by slowing down. Aside from this secondary slowness of speech, there is occasionally also a primary slowness with cerebellar lesions.

*c. Explosive-hesitant Speech:* It is apparently due to a disturbance of the inhibiting and braking function of the cerebellum. The loss of this function results in alternating hypo- and hypermetria of muscle innervations.

*d. Scanning Speech:* It consists of stretching of syllables, which are also being sharply cut off from one another.

The site of the lesion is not quite clearly known, but it seems that scanning speech is due to an interruption of the cerebellar tracts at some point between cerebellum, red nucleus and bulbar centres.

Thus the pathophysiology of the three large motor systems in its relationship to speech illustrates clearly the fact that their intactness and harmonious balance is indispensable for normal speech function. Disturbance in any of the partial functions maintained by these systems throws the entire complex mechanism out of gear and although a certain amount of correction and compensation is possible, physiological conditions never can be reestablished in cases where the damage is irreparable and permanent. Any plan of training for patients with dysarthrias of speech must be conceived with the clear understanding of this situation. Furthermore, the methods applied will have to be carefully adapted to the nature of the motor disturbance that happens to be present.

A few additional theoretical considerations may be helpful in further clarifying the problems before us.

Phylogenetically the pyramidal system is the latest acquisition of the race. Its highly developed and fine performances, such as the use of the hands and of the speech organs, are possible only through the enormous development of the hemispheres of the human brain. It seems further that the frontal lobe is the place where the three large motor systems are coordinated and brought into higher correlations. It is the frontal lobe, according to the theory of H. Hoff's, that establishes new connections for impulses and takes care of their redistribution. It is here where through repression and regulation of primitive functions the fundation for speech development is created. Finally, it is the frontal lobe from where the

connections to the basal ganglia (caudate nucleus) and to the cerebellum lead. Through these connections speech is maintained in a kind of suspension between a conscious and half-conscious state that makes the simultaneous performance of several motor functions possible.

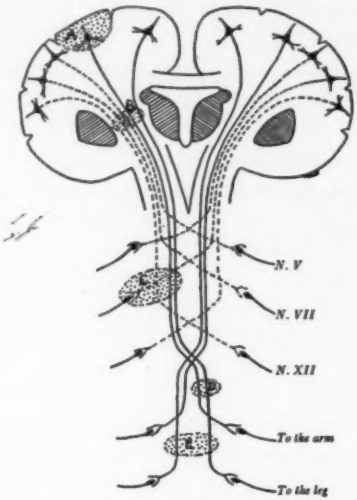



Diagram to illustrate the effects of lesions in various parts of the motor path.

The corticobulbar (pyramidal) system as the developmentally latest part of the motor system is, so to say, superimposed upon the more archaic and more primitive other two systems. This explains why, in case of loss of functions of the pyramidal system, the other two systems may perform fairly completely certain primitive motor functions, while in lesions of these latter systems every kind of pyramidal function will be seriously interfered with.

In other words, the extrapyramidal and cerebellar motor systems may compensate with a fair degree of completeness for the pyramidal system in primitive functions, but not in speech. On the other hand, whenever motor functions of speech served by these two more primitive systems are damaged, there is no possibility of compensation on part of the pyramidal system.

Not only is it impossible for the pyramidal system to compensate for the other two systems, but every normal function in speech of this system is predicated on the undisturbed functioning of the other two systems.

Very much will depend in the process of correction and compensation on residual functions in all three motor systems. More careful analysis of these must form an essential part of any studies in the future.

The psychological concomitant phenomena also will require a great deal of clarification. At present extremely little is known about this side of the problem.

The practical application of the above discussion to the clinical handling of patients must result in the development of a more comprehensive plan of cooperation between various specialties than in the past.

No patient suffering with dysarthria of speech can be handled intelligently without a detailed neurological examination, the purpose of which must be to determine the nature and extent of the motor disturbance and the outlook for compensatory reeducation based on residual functions.

Psychological studies must determine the abilities and disabilities of the patient and must give an evaluation of the total personality. The methods of these studies still await their development and adaptation to the specific conditions of the problem.

Finally, the physiotherapist and orthopedist must correct whatever can be corrected in somatic motor functions.

On such foundations and with clear understanding of the nature of the various disorders present, the educators must develop the methodology that will be adaptable individually to the type of dysarthria that is found in a given patient.

How much may be accomplished with such a plan will be decided only by future experience.

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# THE CLINICAL PICTURE OF DISEASES OF THE LABYRINTH WALL.*†

DR. EDMUND PRINCE FOWLER, New York.

The surgeon must always strive to correlate the clinical with the laboratory and autopsy findings if he expects progress in the understanding of disease. Preliminary to any discussion of pathological material, it is fitting, therefore, to outline the clinical picture. This I shall do in broad strokes, leaving to Prof. Nager and the discussion all pictorial minutiae and controversial points.

The labyrinthine walls are commonly envisioned simply as a bony capsule surrounding the static and auditory membranous labyrinths (some include the internal auditory canal). Besides the portals for the entrance and exit of lymph, blood vessels and nerves, and for pressures and vibrations at the two cochlea windows, the labyrinth walls, like most tissues and all bones, are permeated with spaces for tissue juices, arterioles, veins and neural structures, which largely control their life histories in health and in disease. All the diseases to which other bones are subject may attack the labyrinthine capsule, but all are modified by the unique bone found only in this situation and by the route of invasion from contiguous bone, the middle ear, mastoid, labyrinth and cranial cavities (the latter often through the aqueductus cochleae). The route of invasion may be the sheaths of the nerves, the lymph channels, or the lumen or sheaths of vessels, or all of these pathways.

The labyrinthine capsule normally contains no Haversian systems except in the aged; therefore, circulation is normally almost entirely by diffusion. Basophilic or pseudocartilaginous islands elsewhere found only in the embryo, persist even in the adult human labyrinthine capsule. These peculiarities suggest a lack of at least some rebuilding ability in this tissue and consequent variations in bone pathology.

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*Read before the New York Academy of Medicine, Section on Otolaryngology, Feb. 17, 1937.

†An introduction to the address by Prof. Dr. med. F. R. Nager, of Zurich, Switzerland.

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The signs and symptoms of lesions in the labyrinthine capsule may vary from none to extreme dysfunction, or a total loss of function of the end organs in the membranous labyrinths, or of the facial nerve. Death may and often does result from suppurative diseases attacking the capsule.

Dysfunction must be detected early if one expects to institute preventive measures, and it cannot be too forcibly stated that all medical and surgical measures are in their essence preventive. Heredity plays a major role in many diseases of the capsule, but one must not imagine that this fact alone reveals or explains the cause.

It is known that many lesions of the labyrinthine wall stop at various stages of their development and that some are followed by a repair so complete as to preserve or restore function not only of the walls themselves, but of the structures they encompass.

The clinical picture aside from the personal equation depends upon the extent, virulence and position of the lesions and upon adjacent or distant coincidental or contributing factors. Frequently signs or symptoms which have little or no connection with the labyrinthine wall may simulate symptoms originating therein, and lead one to a false diagnosis. Such symptoms are: Deep-seated pain, diminished hearing or complete deafness, tinnitus, vertigo or other disturbances of equilibrium, nausea, vomiting, positive fistula test, spontaneous or abnormal induced nystagmus and past pointing, and facial paralysis.

Many differing lesions of the labyrinthine wall may give identical symptoms because they are dependent upon interference with the function of the cochlear windows or with the neural mechanism in the static or acoustic labyrinths. The priority and sequence of symptoms depends upon the time and degree of the lesions and the exact place of attack in the conducting and the neural mechanisms of hearing, in the static labyrinth and in the other neural tissues in and about it in the cranium.

One way to differentiate labyrinthine wall lesions is to search elsewhere to discover in other parts of the body similar lesions, especially in the bones of the skull. the ribs, pelvis and the long bones. Search here for evidences of rickets,

osteitis dystrophia fibrosa, osteomalacia, von Recklinghausen's and Paget's disease; also unhealed fractures and new bone formations. (There being no marrow in the capsule, osteomyelitis does not occur, although the bone be contiguous to marrow bone.)

Bone pathology identical with otosclerosis is never found elsewhere than in the hard bony capsule of the labyrinth, and this is one of the things which leads me to believe that otosclerosis is probably not wholly due to a general disease.

Just because some disease is present in the skeleton, it does not necessarily follow that the labyrinthine capsule harbors the same disease or any disease. Contrariwise, because the capsule harbors some disease, it does not follow that it is present elsewhere.

Blood chemistry and cytology may show abnormalities but the ear bones at autopsy show no abnormality. The Wassermann reaction may be 4+ and the ear be normal or harbor some disease other than syphilis, etc. (Avitaminosis and endocrine disorders may be causative factors or merely coincidental.)

Stereoscopic Roentgenograms are a valuable aid to diagnosis. Here again, many misinterpretations are made, because there is so much overlapping of shadows that a disease picture may be shown where there is no disease, or a normal picture in the presence of disease.

Only by employing every means at our command is it possible to approach a correct diagnosis in obscure cases of labyrinthine wall disease.

The correlation of the history and repeated neurological, hearing and labyrinthine tests, with the X-ray and laboratory findings, is the procedure most to be relied upon for diagnosis. Favorable effects from therapy are also of value. A certain diagnosis is obtainable only by biopsy or serial sections at autopsy.

Suppurative diseases involving the labyrinthine walls are usually too extensive to allow of any but radical surgical measures to establish efficient drainage. Here the preservation of function is a secondary matter. It is now sometimes possible by facial nerve grafting to restore function in the VIIth nerve.

Nonsuppurative diseases of the labyrinthine wall are very prevalent, otosclerosis alone being found in at least 5 per cent of autopsies. It is remarkable, in view of the almost universal prevalence of inflammatory episodes in the middle ear and its contiguous spaces, that the labyrinth wall is not more frequently involved in disease. In my opinion lesions of the labyrinthine wall occur (with or without symptoms) in fully 7 per cent of the population reaching middle age. This is a large number. It means that there are in the United States at all times over 9,000,000 persons with some disease of the labyrinthine wall (exclusive of suppurative lesions).

140 East 54th Street.

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## AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Announcement is herewith made by the American Academy of Ophthalmology and Otolaryngology of a change in the 1937 convention site to Chicago, Oct. 10 to 15, 1937; headquarters, Palmer House.

It is with deepest disappointment, the officers, with the approval of the Council, have, because of mechanical difficulties, found it necessary to change the meeting place from Detroit to Chicago.

The Society regrets circumstances developed to prevent honoring the Detroit members by taking the 1937 session to their home city.

# DISEASE OF THE LABYRINTHINE CAPSULE. THE PATHOLOGICAL CHANGES.*

PROF. F. R. NAGER, Zurich, Switzerland.

When these researches were started, one central problem was otosclerosis. As it seemed quite clear that this disease was a bone disease, otosclerosis could only be studied in relation to the general pathology of the bones. Therefore, petrous bones from all known bone diseases were obtained to observe effects of such disease on the labyrinthine capsule.

It is known that the labyrinthine capsule has a special structure, insofar as it consists of three layers: The first being called the endosteal; the second the endochondral; the third the periosteal layer. The second layer contains very characteristic remains of its development throughout life in the form of the so-called interglobular spaces; however, the bone tissue of this layer does not correspond to the thoroughly developed lamellar bone but remains at an inferior stage of development.

We know now that the labyrinthine capsule, therefore, has not only a special histology but even quite a special biology and pathology. This part of the skeleton is the most carefully examined part of human osteology.

The difference between the biology of the labyrinthine capsule and the normal bone lies in the fact that the vital processes of this tissue, known as constant resorption and apposition, are not to be seen in the endochondral layer; whereas the periosteal layer participates in all the well known signs of the vital process in this tissue. The review of general bone diseases, illustrated by a number of lantern slides, may give the following approximate results:

In osteoporosis of old age, where the bone resorption is normal and a normal apposition of bony tissue is deficient, the periphery of the petrous bone looks spongy, whereas the endochondral layer is not affected. The marrow spaces are very large and filled with a fatty marrow.

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Rickets may be observed only in the periosteal layer. In the same way the labyrinthine capsule remains intact in osteomalacia.

On the other hand, in osteolysis, for instance, in consequence of tumors of the VIIIth nerve, *all* bone layers will be destroyed. In carcinoma of the middle ear the same occurs, but the endochondral layer seems to be more resistant.

In suppurations of the middle ear the periosteal layer is very easily destroyed, whereas the endochondral layer shows a larger resistance.

If inflammatory and necrotic processes start in the inner ear, all bone layers may be destroyed, but it will be seen that when the inflammation heals, the bone tissue does not resemble the old endochondral layer. The newly-formed bone shows quite another structure, so that later on, a differentiation between the scar bone and the primary bone is impossible. The hollow spaces of the inner ear are filled up with bone tissue, as, for instance, in deafness after meningitis.

Another characteristic symptom of the bone biology of the endochondral layer is shown by the fact that fractures of the labyrinth do not heal, because the endochondral layer cannot produce a bony tissue; consequently, fractures through the labyrinth remain open, which will be shown by X-ray examinations and which, moreover, may be the cause of a late meningitis.

In Paget's disease the destruction and new formation of bone starts from the periosteal layer. The whole capsule is reformed by unripe bone tissue; the marrow is a fibroblastic one. In advanced stages even the endochondral layer will be affected in the same way, but generally there remains some small portion of the capsule. It is known that in Paget's disease the stapedovestibular joint may be affected, so that the syndrome of ankylosis of the stapes results.

In von Recklinghausen's disease, with tumors of the parathyroid gland, only the periosteal layer was affected in the well known way of formation of fibroblastic bone marrow. Some cases of von Recklinghausen's disease show a very distinct resemblance to otosclerotic tissue, and it might be difficult to differentiate both diseases in very advanced stages.

As to otosclerosis, some typical changes are demonstrated where it is shown that this disease has to be considered as a local osteodystrophy. Details of the otosclerotic alerations are very similar to Paget's and von Recklinghausen's diseases, which confirms the idea of a local osteodystrophy.

In congenital bone disease the labyrinthine capsule shows very typical alterations; in a case with athyreosis the endochondral layer was normal but looked a little underdeveloped; ossification of the periosteal layer was also underdeveloped.

In chondrodystrophy, where the cartilaginous tissue is affected, the ossification seems to be normal with the exception of the ossification zones; they show the same alterations as are known in chondrodystrophy of the extremities.

In osteogenesis imperfecta, the resorption of the cartilaginous tissue is known to be normal, whereas the bone formation is very deficient; this occurs especially in the vestibular part of the labyrinthine capsule, whereas the cochlea remains surrounded by a distinct thin bone layer.

In endemic cretinism, which is known to be frequent in Alpine regions and which leads to dwarfism and other bone deformations, the alterations of the petrous bone are so typical that even without knowing anything else about the case the diagnosis of this disease would be possible from the ear investigation. There is a very marked hyperostosis of the promontory with narrowing of both window niches and certain malformations of the ossicles.

The pathology of the labyrinthine capsule shows many interesting points and, as the result of these researches, it has been found that otosclerosis must be considered as a local osteodystrophy, the last cause of which, we are sorry to say, remains till now unknown.

Freiestrasse 20.

# THE TONSIL.

## PART I. HISTOPATHOLOGICAL STUDIES.

## PART II. THE MUCOUS GLANDS RELATED TO THE TONSIL.

## PART III. THE RELATION OF THE TONSIL TO BRANCHIOGENETIC CYSTS.*†

DR. L. H. MEEKER, New York.

PART I. A conservative estimate based on microscopic studies of abundant material related to the faucial tonsil permits us to say that its pathological variations are as important as any tissues in the human body. The pathology may be considered under the following headings: Development, physiology and disease. The mucous glands and branchiogenic cysts with which the second part of our study is concerned are related to development. The hypertrophies are in part a physiologic response and the diseases include inflammations and tumors. Many excised tonsils show alterations combining these several types. The lesions to be described are best understood by considering the tonsil a varying deposit of lymphoid tissue at the inner opening of a diverticulum of the pharynx,[1] representing the second branchial cleft. The diverticulum is usually closed except at its inner end and here represents a shallow cleft which may, however, be patent from the supratonsillar fossa to the side of the neck, usually at the anterior border of the sternomastoid muscle.[2] The area covered by surface and crypt epithelium for both tonsils, based on the average size for hypertrophied tonsils for all ages, is approximately 6x6 cm., or roughly the size of the palm of the hand.

Inflammations of the tonsil are designated as lacunar, parenchymatous, and peritonsillar, acute or chronic.[3] We must consider the component tissues of the human tonsil: The epithelium, the lymphoid tissue or parenchyma and the

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†From the Department of Pathology and Bacteriology, New York Post-Graduate Medical School and Hospital, Columbia University.

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environment or tonsillar bed with its fascia, muscle and mucous glands.

The crypt content varies greatly and may be filled with mucus or cellular exudate consisting of epithelium, lymphocytes and polymorphonuclear leukocytes. Exfoliated epithelium is common, and if a crypt is plugged and distended, it may be filled with epithelium which has undergone degeneration with the formation of cholesterol crystals, cholesteatoma.[4] Bacteria are abundant in the crypt contents and vary from those usually found in the mouth to abundant colonies of

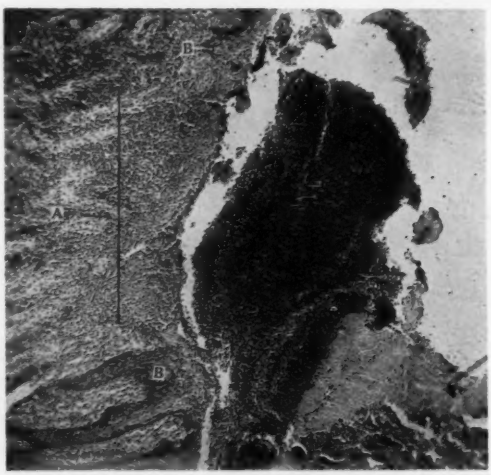


Fig. 1. F. 40 years. (A) Acute ulcer engrafted on chronic recurrent tonsillitis. Marked hypertrophy. (B) Crypt epithelium at margin of ulcer.

vibriothrix.[5] Another common fungus is the so-called mycosis pharyngis, which lodges in the rough cornified epithelium of parakeratosis but does not initiate the keratinization.[6,4] Foreign bodies are sometimes found in the crypts, food in particular. Occasionally stones, "tonsilloliths," are present in the crypts and these are formed almost invariably about the vibriothrix colonies.

The crypt epithelium presents a variety of pathological changes. It is frequently thickened and vesicular with polymorphonuclear leukocytes in the vesicles. These vesicles may

rupture and provide an entrance for bacteria present in the crypts and so produce ulceration. In the systematic examination of tonsils small ulcers are a common finding and are often seen in the hypertrophied tonsil. These may be single or multiple and they may be found in an acute (see Fig. 1) or in a healing stage (see Fig. 2). Shallow ulcers may heal or the inflammation may extend into the underlying lymphoid tissue. Adhesions may form in the crypt walls and the contained tissue detritus with cholesterol crystals and calcific deposits may cause a foreign body reaction.[4] Not infrequently such crypt walls are replaced by granulation tissue and later

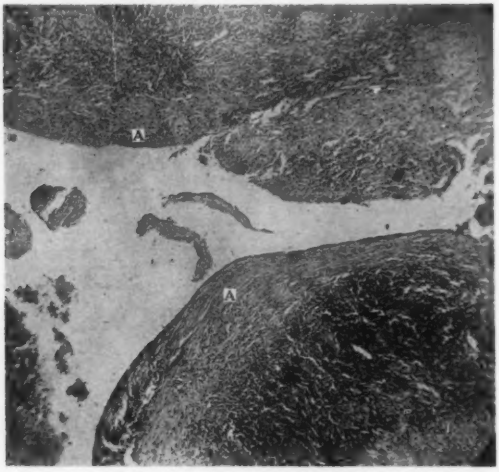


Fig. 2. M. 13 years. (A) Scars in crypts, healing stages of ulcers. Hypertrophy.

fibrous tissue, even with extensive fatty or xanthomatous degeneration.[4] The parenchyma is usually involved by extension from the crypt walls. Occasional microscopic abscesses are found in the follicles of the parenchyma, which may be hematogenous, and bacteria may be found in the necrotic focus.[4] Many tonsils show a combination picture of extensive granulation tissue replacement of parenchyma as well as fibrosis, the picture varying in different areas of the same tonsil. Changes seen in the lymph follicles, such as abundant large mononuclear cells[5] demonstrated in measles and typhoid fever, are chiefly of histological interest.

Peritonsillar abscesses may invade the peritonsillar fascia directly from the crypt inflammation (see Fig. 3). The septa and peritonsillar fascia, commonly called the capsule, often shows increase of fibrous tissue, the fibrous tissue of the septa invading the lymphoid tissue and that of the fascia invading the underlying muscle with fixation of the tonsillar mass. In this fibrous tissue we frequently find a varying cellular exudate, acute or chronic type, about the blood vessels, the reaction often severe enough to obliterate even the larger vessels, or extend into and about the mucous gland ducts. The peritonsillar fibrosis interferes with the function of the embedded

Fig. 3. M. 12 years. (A) Peritonsillar abscess (accidental finding). (B) Tonsil-hypertrophy.

mucous glands and prevents the proper evacuation of the crypt contents.[7]

Hypertrophy of the tonsil is a relative term depending upon the age of the patient. Fifty per cent of such hypertrophies, especially in youth, are said to be due to heredity. The enlarged or hypertrophied tonsil comes so frequently to the pathologist that it is possible to recognize the importance of recurrent inflammation. This is the type invariably seen in adults and a more common cause of hypertrophy than so-called status lymphaticus. When viewed from the histopathological picture presented, the hypertrophied tonsil usually discloses some or all of the conditions enumerated, in addition

to the enlarged follicles and their multiplication. The same cause may result in later atrophy, even in childhood.

Tuberculosis of the tonsil may be diagnosed clinically, but it has more often been an accidental microscopic finding. There may be a few tubercles or the process may be diffuse and involve one or both tonsils. Ulcers in the crypts due to tuberculosis are rare. Usually there are tubercles buried in the parenchyma and for these a hematogenous origin seems probable. Not infrequently tuberculous lymph nodes of the neck prompt an investigation of the tonsils. We have not encountered syphilitic tonsillitis of the primary type and other syphilitic lesions have been extremely rare.

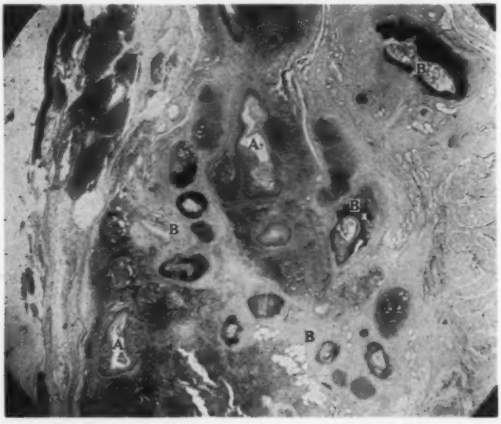


Fig. 4. M. 35 years. (A) Crypts. (B) Cartilage and bone with fat in marrow spaces (28 islands in one section). Secondary tonsillectomy.

Parasites found in the tonsil have been reported in a few instances, among them the round worm.[8] Trichinae have been found in the muscle at the base of the tonsil, and we are convinced that the pharyngeal muscles are a favorite site for the encysted forms.[4] Four such cases were encountered in an epidemic in New York City in the spring of 1929.

"Tonsilloliths" deserve special mention. They are usually small and lie free in the crypts. Recent reports include one weighing an ounce and another measuring 8x6 mm. They are composed of cholesterol, calcium and phosphates in vari-

ous combinations.[4] In the septa and pharyngeal fascia the deposits of cartilage or, more particularly, of the bone have been called "tonsilloliths."[5] Another interpretation is to be preferred and here there are two points of view: One, that they are the result of chronic inflammation, derived from

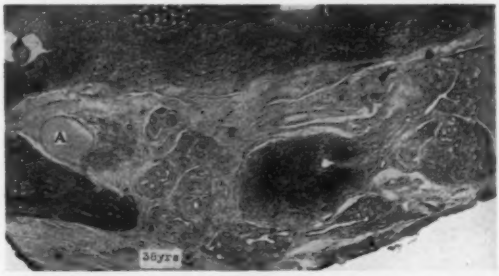


Fig. 5. F. 38 years. (A) Fetal cartilage. (B) Hyperplastic mucous glands. Peritonsillar abscesses for three years.

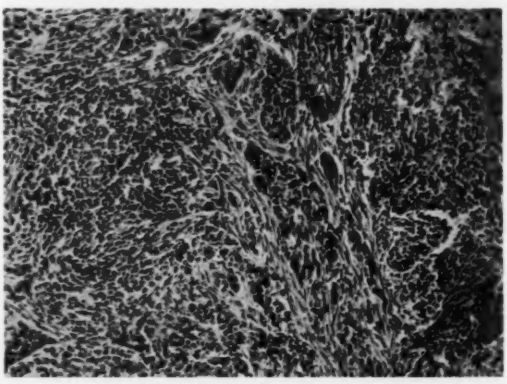

Fig. 6. M. 48 years. Lymphoepithelioma. (A) Groups of epithelial cells at right. (B) Mixed with lymphocytes at left.

multipotent mesodermal cell rests (see Fig. 4); another view is that of persistent branchial arch remnants. One type that we have encountered upholds the second explanation because it has occurred repeatedly at one pole of the tonsil and in one site with associated hypertrophic mucous glands (see Fig. 5).

There is a great variety of neoplasms of the tonsil, both primary and secondary. The primary tumors include the leukemias, fibromas, fibrolipomas, angiofibromas, angiomas, psammoma, reticuloma, papilloma and dermoid cysts among the benign types. To this list we may add mixed tumors of salivary gland and plexiform neuroma from our own series.[10]

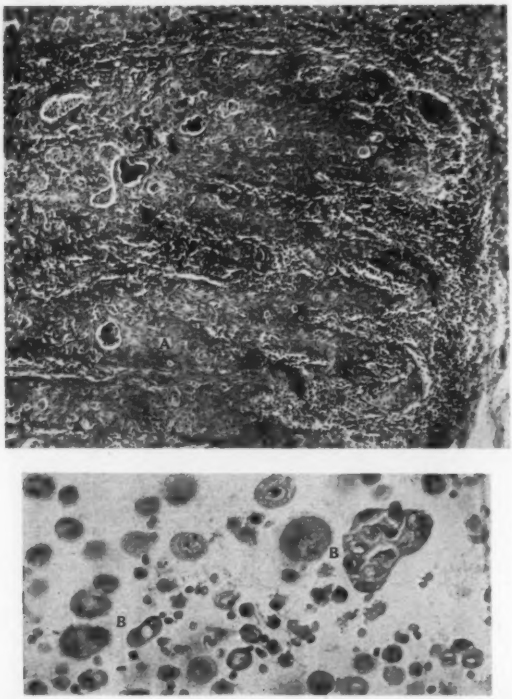


Fig. 7. M. 50 years. Epidermoid carcinoma (accidental finding). (A) Bizarre growth of epithelial cells. (B) Metastatic cells in pleuritic fluid following metastasis to lungs (two months after tonsillectomy).

The malignant types include lymphosarcoma, lymphoepithelioma (see Fig. 6) and squamous cell carcinoma (see Fig. 7). The secondary tumors, other than those invading the tonsil by continuity, are metastatic carcinoma from the breast, the testicle, the lung, the colon and sarcoma from the periosteum, as well as metastatic hypernephroma and glioma. On the other

hand, tumors of the tonsil may metastasize to local lymph nodes and produce distant metastases, as the lung (see Fig. 7B), the chief offender in this group being the lympho-epithelioma.

PART II. Beneath the faucial tonsil and intimately related are the mucous glands and branchiogenic cysts to be described. We have found that the mucous glands related to the tonsils are subject to wide variation, both in location and manner of opening to the surface. The usual location of these glands is in the tissues at the periphery of the tonsillar mass with ducts opening into adjacent crypts. There is to be noted

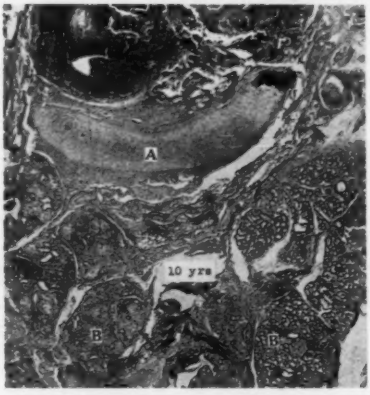


Fig. 8. F. 10 years. (A) Fetal type cartilage bar. (B) Associated hyperplastic mucous glands.

among these a frequently recurring group of mucous glands situated in the "hour-glass" constriction present between the posterior and anterior anlagen of the tonsil in the fetus. These are unusually large glands and are often associated with an island of cartilage and both may be embedded within the tonsillopharyngeus muscle. The cartilage is of interest chiefly because it may be considered a remnant of the fetal cartilage which early encloses the entire tonsil area (see Fig. 8). This is the view held by Dietrich,[11] and Jordan and Kindred[12] refer to an "ancestral remembrance," calling attention to the tonsils of the horse, dog and cat. The tonsils of these animals are embedded in a recess of the epihyal cartilage. The pres-

ence of this fetal cartilage is significant when added to other evidence of the instability of this area.

A central location of the mucous glands has been denied by many authors, most recently by Tramontano[13] and by Silvagni.[14] Our own observations have shown there are many mucous glands located beneath the central portions of the

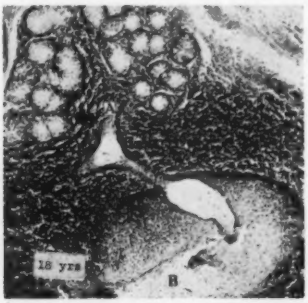


Fig. 9. F. 18 years. (A) Mucous gland ducts with squamous epithelium emptying into (B) crypt. Central position.

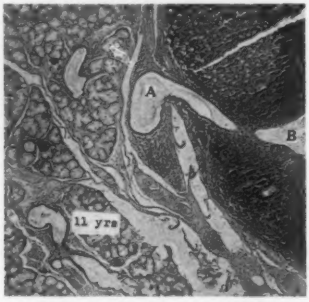


Fig. 10. M. 11 years. (A) Mucous gland with columnar epithelium emptying into (B) crypt. Central position.

tonsil (see Figs. 9, 10 and 11). We are chiefly concerned in demonstrating the glands of this particular area and their relation to tonsil pathology.

The facts brought out by recent studies of Fox,[15] Grünwald,[16] Goerke,[17] Kingsbury[18] and Levin[19] added to the controversial evidence of previous workers, as Hammar,[1] Levin-

stein[20] and Maximow,[21] force us to the conclusion that the tonsillar area in its development is the most unstable portion of the second pharyngeal pouch, as affirmed by Ewing.[22]

The distribution of mucous glands related to the tonsils is an illustration of this instability. The human tonsil develops in various directions related to the primary anlagen and duplicates one or another mammalian type. The mammalian types differ widely, as shown by Grünwald, Seccombe, Hett and Butterfield.[23] There are tonsils with very few associated

Fig. 11. F. 26 years. (A) Mucous glands beneath centre of tonsil. (B) Crypts. History of chronic peritonsillitis.

mucous glands and tonsils resting on a continuous mucous gland bed. Some mammals have no tonsils, as the mouse, rat and guinea pig, and some have no mucous glands in this region, as the domestic pig.

The type of epithelium found where mucous glands open into the ducts varies. Ducts lined by squamous epithelium may extend to the squamous epithelium of the crypts (see Fig. 9) and the ducts may be single or in groups. Ducts may

also be lined by cuboidal (see Fig. 10) or columnar epithelium and the epithelium may be ciliated. This agrees with descriptions of the ducts of the mucous glands of the pharynx in general. The ciliated columnar epithelium is of most interest, especially in the group of glands situated centrally beneath the middle lobe of the tonsil. Here there is no possibility that lymphoid tissue has extended to include the ciliated columnar epithelium of the adjacent pharyngeal mucous membrane.

Ciliated epithelium found in the fundi of centrally located crypts is accepted by Dietrich for the first few years of life and he considers it a fetal remnant. He mentions that he has seen it *once* in the tonsil of an adult. We have found it many times in the tonsillar crypts of adults and consider it

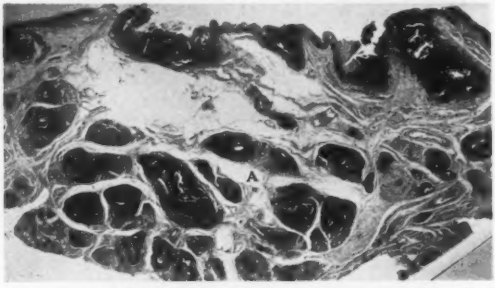


Fig. 12. M. 49 years. (A) Lymphoid tissue about mucous glands. (B) Primary tonsil site (secondary tonsillectomy). History of "quinsy" three times and enlarged cervical glands.

a fetal remnant since there are occasionally other associated fetal anomalies as persistent branchial clefts.[24]

It may be said in general that ciliated epithelium in the fundus of a crypt indicates the opening of the duct of a nearby mucous gland or embryonal cyst. The area lined by ciliated columnar epithelium is rarely more than a millimeter or two in diameter. Such ciliated epithelium does occur without relation to mucous glands or cysts and is undoubtedly a persistent fetal remnant.

Partial studies have been made by Rauber and Kopsch,[25] Barnes,[26] Seccombe, Hett and Butterfield, Silvagni and others, but there is no published detailed study of mucous glands associated with the tonsils in spite of the fact that recent authors have indicated an interest in the subject. Neumann[27]

and Syk[28] questioned clinically whether mucous glands could be the site of peritonsillar abscesses. Kingsbury considered the possibility of the ducts of mucous glands being transformed into tonsillar crypts and Levin asks whether lymphoid tissue is inimical to the growth of mucous glands.

We have examined hundreds of tonsils cut in both longitudinal and transverse planes. Many serial sections have been made. The findings in longitudinal sections agree with those of most observers. Transverse sections have yielded the most information about the unusual locations of mucous glands and their manner of opening on the surface. In addition to the variations already noted we have occasionally found glands

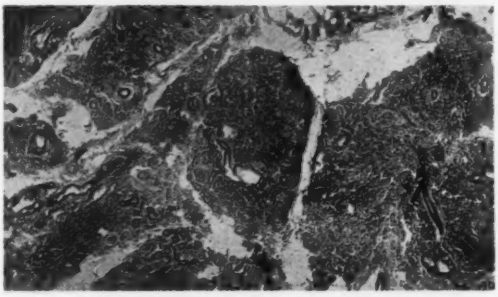

Fig. 13. High power of (A) in Fig. 12.

10 to 15 mm. in diameter and a few glands wholly or in part duplicating the parotid gland in structure.

The anatomical differences noted assist us in understanding tonsil pathology. This is especially true of the so-called "recurrent tonsil" of the adult and of peritonsillar abscess. We have found true lymphoid tissue extending along mucous glands deep into the pharyngeal fascia (see Fig. 11) and muscle and not extending along blood vessels. Grünwald approximates this description and Seccombe, Hett and Butterfield describe extensions of lymph follicles about mucous glands in the gorilla and Vervet monkey. This extratonsillar lymphoid tissue may proliferate extensively after excision of the tonsil proper (see Figs. 12 and 13). Peritonsillar abscesses may arise in this periglandular lymphoid tissue (see Figs. 14A and B).

PART III. Cysts of the neck have been variously designated depending upon the interpretation of different authors. There is objection to the term "branchiogenetic" for the reason that the human embryo at any stage does not possess struc-

Fig. 14. (A) M. 28 years. Peritonsillar abscess about necrotic mucous glands. Secondary tonsillectomy.

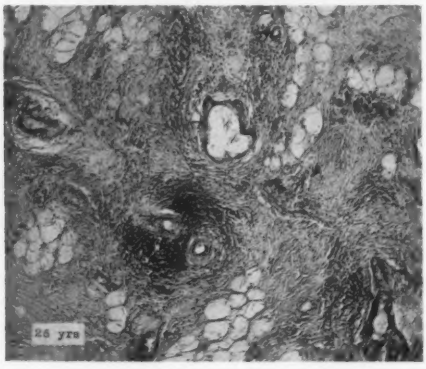


Fig. 14. (B) F. 26 years. Scar tissue about mucous glands. History of previous peritonsillar abscesses.

tures that may properly be considered the precursors of congenital cysts of the neck. It is necessary to consider the homologous organs of lower animals in order to interpret the findings in many instances of human maldevelopment.

We are confronted with this problem in some of the cysts we are about to discuss. Many of the cysts of the neck and in particular those near the angle of the jaw are tonsillar in origin. They are characterized by an abundance of true lymphoid tissue in the walls lined by squamous epithelium.

The first observation in regard to these cysts was made by Lucke,[29] 1861. His diagnosis was that of an epithelial rest in a cervical lymph node, the fetal rest of Cohnheim. Albarran[30] called them cystoepitheliolymphoides. Later, Schede,[31] Neumann and Baumgarten[32] and Albarran reported findings agreeing with Lucke and made the same diagnosis. Darrier,[33]

Fig. 15. (A) 38 years. Wall of branchial cyst of neck "type amygdallien." The nodules are lymphoid tissue.

1894, saw a fistula with a similar lining and was the first to consider the relation of these cysts to the pharynx. Bottini[34] demonstrated such a cyst continuous with a fistula leading to the pharynx and also claimed that all similar cysts of the neck at one time have a duct communicating with the pharynx. Stephan agreed with this view.

It remained for Terrier and Lecene,[35] 1905, to collect the reported cysts of this type and to add two of their own. They suggested the term "type amygdallien" (see Figs. 15). Cotelloni[36] agreed that they were quite distinct from the dermoid cysts with which they had previously been classed because of their lining of squamous epithelium. Albrecht reported cysts

having true lymphoid tissue in the walls but lined by cylindrical epithelium. Cysts of this type with papillary formations have been reported by Peyron,[37] Houdart,[38] Hufnagel[39] and Warthin.[40] Peyron and Rouis la Croix[41] found bodies resembling Hassal's corpuscles in cysts with lymphoid tissue walls. These they designated "types Hassalliens." Frazer[42] explained them as a "philogenetic memory" of the subdermal thymus of the neck seen in the mud eel.

We are attempting to show that any of these types may arise in the tonsillar region (see Figs. 15B and C). The embryological hypothesis explaining these cysts is substantiated by associated fistulae[43] leading to the supratonsillar fossa which is a common finding in relation to these cysts

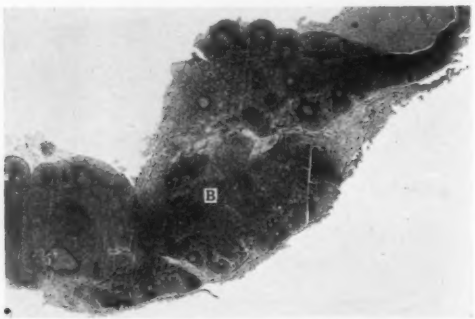


Fig. 15. (B) Low power of microscopic section of wall. The lymph follicles are numerous.

(see Fig. 16). According to Rabl,[44] the fistulas lead to some persistent portion of the precervical sinus related to the second pharyngeal pouch. Winglowski[45] claimed the origin was in some portion of the thymopharyngeal duct. These hypotheses have been gradually discarded in favor of the more detailed descriptions of the developing pharynx in lower animals and man.

According to Kingsbury the upper lobe of the tonsil develops in the second pharyngeal pouch and a lower lobe from an adjacent area in the third pharyngeal pouch closely related to the indefinite branchiomeres from which the thymus may or may not develop. It appears that the thymus has no

embryonal placodes and many develop in the ventral portion of any pharyngeal cleft.[45]

Grünwald derives the tonsil from what he designates a "thymic metamer" in the ventral pocket of the second pouch, the area in which the thymus is developed in some of the lower animals as, for example, the rabbit. Kolliker,[46] Stöhr,[47] Piersol[48] and Verdun[49] had previously made this observation. These findings serve to indicate the general instability of the tonsillar region.

In the study of the relation of mucous glands to the tonsil we demonstrated columnar epithelium both with and without cilia, in the fundi of crypts. In some instances these were in areas where mucous gland ducts opened into the crypts (see

Fig. 15. (C) High power of (B). The squamous epithelium, lymph follicles and crypt are typical.

Figs. 9 and 10). In other cases there were no related mucous glands. Instead we were able to demonstrate associated minor branchiogenic clefts (see Fig. 16). Deeply placed squamous epithelium beneath the tonsillar parenchyma, even between the muscle bundles, has not been up to the present considered significant but must be regarded as related to branchiogenic cysts. Cysts lined by squamous epithelium with walls of lymphoid tissue constitute a well recognized group generally accepted as being of tonsillar origin (see Figs. 15 A, B and C). The French have written most upon this subject, as in the collected cases of Thomann,[50] 1925, and Moatti,[51] 1929. Moat-

ti's series included three examples of "cystes amygdalliens" which he said resembled both tonsil and thymus.

Cysts with walls of true lymphoid tissue lined in part by columnar epithelium and in part by squamous epithelium or by ciliated columnar epithelium may also be considered as of tonsillar origin in some instances.

Such cysts are included in Nylander's[52] collected series, 1928. The cyst of his Case 26, lined in part by squamous and in part by ciliated columnar epithelium, communicated with the tonsil by a fistula. Case 20 also showed a fistula extending to the tonsil, lined by ciliated epithelium. Stephan[53] also found fistulas reaching the tonsil. One of our minor branchial cysts was related to a fistula of one side extending from the tonsil

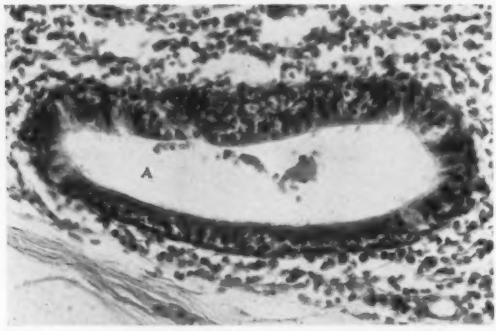


Fig. 16. M. 50 years. (A) Opening of minor branchiogenic cleft at base of tonsil crypt. (B) Lining of ciliated columnar epithelium (high power). Other fetal anomalies as ultimo branchial bodies in hypertrophied thyroid, Riedel's struma.

(see Fig. 17) to a cleft behind the ear. Sercer's[54] report describes absence of ears and coincident absence of tonsils, another example of such associated anomalies. Precechtel[55] reported familial developmental defects. Seven members in three generations of the same family had either congenital fistulas of the neck, diverticula of the pharynx or branchial cysts.

## SUMMARY.

*1.* Hypertrophy of the tonsil is usually the result of chronic recurrent tonsillitis.

*2.* Cartilage and bone are due to fetal rests in many instances.

*3.* Mucous glands open into crypts and are closely related to tonsil pathology, *i. e.*, peritonsillar abscesses.

*4.* The true lymphoid tissue found beneath the main tonsillar mass in the adult is an extension along mucous glands (not along blood vessels). The recurrent tonsil in the adult arises from residual tissue about the mucous glands.

*5.* There is a constantly recurring group of mucous glands associated with a bar of cartilage. These are always in the same location, in the constriction between the middle and lower lobes of the tonsil.

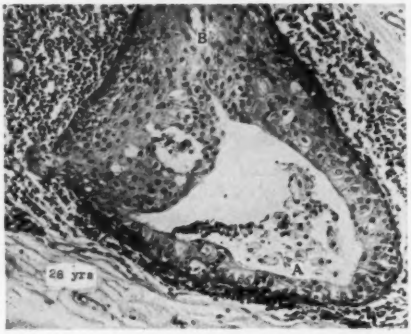


Fig. 17. M. 28 years. Embryonal defect at base of tonsil communicating with cleft behind ear. (A) Fistula lined by columnar ciliated epithelium. (B) Tonsil parenchyma (high power). (Branchial clefts behind both ears.)

*6.* Ciliated columnar epithelium in the fundus of a crypt related to the central tonsillar mass is to be considered a fetal remnant. It may be related to mucous glands or to minor branchiogenic clefts.

*7.* Many cysts of the neck have walls of true lymphoid tissue and are lined by squamous epithelium, denoting their tonsillar origin.

Finally, to quote Kingsbury, "The current statements of the development of the tonsil are inadequate."

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# THE DISAPPEARANCE-TRENDS OF STAMMERING: A PRELIMINARY REPORT.

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When measured, objective data become available about an abstract problem; usually they tend to promote a better understanding of it. This appears to be true, for example, concerning the problem as to the behavior of the unknown factors which determine a person's "handedness" status. Now two kinds of fairly exact objective data can be obtained from handwriting which seem to bear upon this problem. They consist, respectively, of graphic lineage measurements and graphic time measurements.

In regard to the lineage data, it is a simple matter to obtain these with a millimeter rule; but, for the purpose in hand, the only useful values of this sort are those measured on samples of bimanual handwriting. Experience has shown that any literate person can produce this type of script simply by setting down a model test word — *e. g.*, the word "Halliard"— with both hands at once, each hand moving in the direction away from the midline of his body. Of course, each specimen such as this exhibits two bits of scripts; and almost always these two graphic efforts differ from one another as to length. Now in an earlier study[1] samples of two-handed penmanship having been obtained from 100 persons — 84 dextrals and 16 sinistrals — and the lineage data having been duly noted on each one, I found that 71.66 per cent of the dextrals had produced the larger values with the left hand while, on the contrary, 90 per cent of the sinistrals had run up the larger values with the right hand. Without exception the two bits of writing thus thrown off sprawled irregularly along the baseline of the text. Seemingly, therefore, the central factor which had caused the muscles of the upper extremities to exhibit this sort of stammering activity was, presumably, the precise synchronization of the two cerebral motor discharges concerned — *i. e.*, left and right. Although it was assumed that two kinetic forces of unequal intensity

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had reacted upon one another and thus in some way disorganized the handwriting movements, it did not seem practicable by means of graphic lineage data to elucidate further the question as to the nature of the sprawl phenomenon. However, with the aid of graphic time data some definite progress was made in this direction.

Having convinced myself in collateral experiments that any educated person can write with each hand in the direction either dextrad or sinistrad, in a later research[2] exact time values as regarded the four handwriting activities thus possible were secured from four persons — four dextrals and two sinistrals — by means of a stopwatch and a model sentence.

In this study a single round of time measurements was made once daily with each person tested; and this work was repeated on 25 nearly consecutive experiment days. It should be stated that none of these reactors did any writing practice whatever.

Naturally I obtained in this way from each person tested exact time values as regarded two bits of reversed writing — one set down with his right hand, the other with his left hand. On tabulating side by side in two separate panels the mirror-writing values obtained, respectively, from the dextrals and from the sinistrals, it became obvious at once that the time data displayed clearly two remarkable phenomena, depending upon the point of view.

Thus if observed in the direction from above downward, each column of time values exhibited strikingly a well marked process of progressive acceleration since, in that direction, the figures became smaller at a fairly regular rate. This evidence seemed to show that a central motor impulse on being switched into a new transmission line tends to traverse it with ever increasing velocity.

On the other hand, if the mirror-writing time data of the two dextrals — *i. e.*, in each instance a right value and a left value — were compared on any horizontal line with the corresponding data as to the two sinistrals, it was noted invariably that the values of the dextrals diminished from left to right, while those of the sinistrals diminished from right to left. Hence the data arrayed in the two panels exhibited a curious phenomenon of isomorphism. In this respect, then, the time

data agreed with the lineage data. Since, however, a given line may be produced by a force which moves no faster than a glacier or, again, by the tangential impact of a projectile, from a practical point of view the kinetic data would seem to be of the major interest. On the whole, therefore, such graphic data as I had showed that laterality effects manifest themselves distinctly in suitable specimens of handwriting.

These experimental tamperings with the handedness factor having in no instance caused the slightest disturbance of the speech faculty, and suitable material being available, experimental studies were set on foot with two stammerers — M. S., a girl, age 17 years, and H. A., a woman, age 35 years. On undertaking this work, the purpose in view was to find out whether their respective central nervous kinetic impulses into new channels, as in the performance of the aforementioned unwonted types of handwriting, would in any way modify their episodes of stammering. Since the results obtained respectively from these two reactors agreed almost perfectly, I shall describe only those referable to M. S.

The experimental plan pursued was to collect once daily, by means of a stop watch and a model sentence, four timed specimens of unimanual handwriting — *i. e.*, with each hand, one written dextrad, the other sinistrad. This work was repeated on 25 as nearly as possible consecutive experiment days.

After the time tests had been in progress for one week, and for approximately three months thereafter, at irregular intervals speech tests were made in such a manner as to yield quantitative results. To this end on any given day the reactor pronounced four times in succession a panel of 25 words, the errors committed on each panel being noted separately. In this way a percentage figure became available. On comparing from day to day the successive figures referable to each panel, it was noted at once: *1.* That the speech errors made on any given day tended to pinch out more or less rapidly in the direction from left to right; *2.* that eventually, if the same words were repeated on successive days, the stammering ceased completely and abruptly.

The object of this article is to describe briefly these seemingly characteristic disappearance-trends.

## METHOD.

It should be stated at first, incidentally, that in preliminary experiments made in the manner already described four unimanual samples of timed writings were obtained from 27 stammerers — 21 dextrals and six sinistrals. Also from two of the dextrals successive panel values were secured with respect to faults of pronunciation; but the data obtained from them in regard to their graphic time values were inadequate for the determination of the brainedness factor.

TABLE 1.

LISTS OF WORDS USED IN THE PANEL TESTS.

| 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|
| *Downey | Downey | Disestablish | Whittle | Mower | Zebulon |
| Business | Business | Domesticity | Thunder | Hemlock | Millet |
| Helena | Riot | Butchery | Smear | Nuncio | Gurgle |
| *Bobby | *Mother | *Motherhood | Mutter | Murmur | Mammal |
| Jacobs | Submit | *Hemisphere | *Nutrition | Native | Yataghan |
| Vienna | Industry | Bobby | Whistle | Typical | Vehement |
| *Tulip | *Disposition | Number | Thrift | Mottle | Ultimate |
| *Chopper | *Wilson | Wilson | Shave | Whittle | Querulous |
| Emma | Invested | Invested | *Mugger | Notation | Ribbon |
| Bologna | Goggle | Mrs. | Nifty | *Mosquito | *Omnivorous |
| *Rogers | Production | Protuberant | Whisper | Newtown | Nullify |
| *Figure | *Mrs. | Theater | Thrive | *Mullet | Hummock |
| Jelly | Delivering | Gutteral | *Mummy | Muddle | Minimal |
| *Garage | Theater | Nature | Nymph | Butter | Immunity |
| *Petunia | William | Whither | Whoop | Bitter | Lullaby |
| *Tuttle | *Motion | Motion | Thyroid | *Mister | Lubbock |
| Junket | Ruin | Ruin | Shatter | Mortar | Maple |
| Whippet | Gutter | Washing | *Mulatto | Moulting | Numeral |
| Senator | Must | Wearisome | *Numeral | Nickname | Vivify |
| Rowett | Watching | Simian | Whither | Moslem | Mummy |
| *Bigelow | *Number | *Noontide | Thrips | Mumpish | Whiffle |
| Salmon | Supervisor | Siphon | Shamble | Skewer | Naples |
| *Nugget | *Senior | *Moonlight | *Multiply | Muscovite | Muffle |
| *Canary | *Nature | Rummage | Norwich | Mulberry | Money |
| Billy | Site | *Money | Shoot | *Mimeograph | Mugger |

N. B. Only those words missed in the first panel are starred.

To remedy this deficiency, in the present research the necessary quantitative data were secured from two stammerers, both as regards the time factor and as to the number of speech errors made. Hence I had four different sets of panel data; and it could be seen at a glance that all four exhibited the disappearance-trends. The findings referable to M. S. were typical of this group.

With the exception of her stammering, which is not particularly noticeable, in the physical sense M. S. is a normal,

vigorous girl, decidedly athletic in type. She is a popular student in the high school, and she is forward in her studies. Her chief complaint about the speech disorder is that it is

TABLE 2.

SPEECH ERRORS MADE IN THE FOUR PANELS.

| | PANELS: | | | |
|---|---|---|---|---|
| | 1 | 2 | 3 | 4 |
| | Downey | Downey | | |
| | Bobby | Helena | Helena | Bobby |
| | Tulip | | Tulip | Tulip |
| | Chopper | Chopper | Chopper | |
| | Rogers | | | |
| List 1 | Figure | Figure | Figure | |
| 8/5 | Garage | Garage | | |
| | Petunia | Petunia | | |
| | Tuttle | | Tuttle | Tuttle |
| | Bigelow | Bigelow | | Bigelow |
| | Nugget | | Nugget | |
| | Canary | Canary | Canary | Canary |
| | Mother | Downey | Riot | |
| | Disposition | Business | Mother | |
| | Wilson | Mother | Wilson | |
| List 2 | Mrs. | Invested | Production | |
| 8/8 | Motion | Theater | Motion | |
| | Number | William | Number | |
| | Senior | Ruin | Senior | |
| | Nature | Watching | | |
| | Motherhood | Domesticity | | |
| List 3 | Hemisphere | Bobby | | |
| 8/15 | Noontide | Mrs. | | |
| | Moonlight | | | |
| | Money | | | |
| | Nutrition | | | |
| | Mugger | | | |
| List 4 | Mummy | Mummy | Mummy | |
| 9/5 | Mulatto | Mulatto | Mulatto | Mulatto |
| | Numeral | | | |
| | Multiply | | | |
| | Mosquito | Mosquito | | |
| List 5 | Mullet | Muddle | | |
| 9/15 | Mister | Mister | Mister | |
| | Mimeograph | Moslem | Mimeograph | |
| | | Mimeograph | | |
| List 6 | Omnivorous | Minimal | Omnivorous | |
| 11/10 | | | Money | |

prone to annoy her, especially when she is called upon to introduce people to one another or to stand up in school to recite.

Once daily she was timed with a stop watch while she wrote with each hand — dextrad and also sinistrad — this sentence: "He protested that he couldn't eat the twenty-two tarts; and the truth is that he ate twenty."

The errors she made in pronunciation were noted down in her own home, in the presence of an audience consisting of the members of her family. One of these, who acted as her helper, slowly read aloud the list of test words and M. S. tried

TABLE 3.

THE DISAPPEARANCE-TRENDS OF STAMMERING. THE CONSECUTIVE PANEL VALUES SHOW THE NUMBER OF SPEECH ERRORS MADE ON REPEATING FOUR TIMES A LIST OF 25 WORDS.

| | Date | Test List | PANELS: 1 | 2 | 3 | 4 | Speech Errors Per Cent |
|---|---|---|---|---|---|---|---|
| | 8/5 | 1 | 12 | 8 | 7 | 5 | 32 |
| | 8/8 | 2 | 8 | 8 | 7 | 0 | 23 |
| | 8/12 | 2 | 0 | 0 | 0 | 0 | 0 |
| | 8/15 | 3 | 5 | 3 | 0 | 0 | 8 |
| Vacation | 8/25 | 3 | 5 | 1 | 0 | 1 | 7 |
| | 8/29 | 3 | 2 | 1 | 0 | 0 | 3 |
| | 9/5 | 4 | 6 | 2 | 2 | 1 | 11 |
| | 9/6 | 4 | 0 | 0 | 0 | 0 | 0 |
| | 9/19 | 5 | 4 | 5 | 2 | 0 | 11 |
| | 9/20 | 5 | 1 | 3 | 1 | 0 | 5 |
| | 9/21 | 5 | 3 | 1 | 1 | 0 | 5 |
| | 9/22 | 5 | 2 | 1 | 0 | 0 | 3 |
| | 9/23 | 5 | 2 | 1 | 0 | 0 | 3 |
| | 9/24 | 5 | 0 | 0 | 0 | 0 | 0 |
| In School | 10/3 | 5 | 0 | 0 | 0 | 0 | 0 |
| | 11/10 | 6 | 1 | 1 | 2 | 0 | 4 |
| | 11/11 | 6 | 4 | 2 | 4 | 2 | 12 |
| | 11/12 | 6 | 4 | 3 | 2 | 1 | 10 |
| | 11/13 | 6 | 5 | 3 | 1 | 0 | 9 |
| | 11/14 | 6 | 0 | 0 | 0 | 0 | 0 |
| Totals | | | 64 | 43 | 29 | 10 | |

her best to pronounce each one. The question as to the score to be set down opposite each word spoken was decided by M. S. herself; and she had been instructed to record as an error the slightest repetition of a sound or blocking on a syllable.

Although M. S. stammered when she tried to pronounce various words, her chief difficulty concerned the nasal tones "M" and "N." For example, the enunciation of the word

"mother" or the word "Mrs." was often a sore trial to her; and she was apt to render "number" as "thumber." Consequently, in setting up the lists of test words (see Table 1). to be used in the panel tests, the principle followed was to select those which it seemed likely would provoke stammering. Thus it will be noted in the test lists from No. 1 to No. 6 that the tone "M" occurs respectively two, seven, 11, 16 and 14 times.

## RESULTS.

The figures displayed in Table 3 speak for themselves, since they show the disappearance-trends distinctly. However, a few comments seem necessary. On referring to this table it will be noted: *1.* In the horizontal direction, from left to right, that the panel data tend to pinch out at each level; *2.* in the vertical direction, from above downward, that after a variable number of days the stammering ceased completely and abruptly. However, there was a considerable difference between the lists — for example, between List 4 and List 5 — as regarded the time element, the former clearing on the second, the latter on the sixth day. What caused this difference? It seems possible, or at least it may be suggested, that the factor which caused the stammer to persist so long — as regarded both List 5 and List 6 — was the factor of nervous tension set up by her school activities.

Although the data shown in Table 2 are incomplete as regards Lists 5 and 6, they enable one to compare the disappearance-trends with the various words which cause articulation difficulties.

Did the performance of the unwonted handwriting movements modify in any way the tendency toward stammering? On this point the evidence obtained was somewhat inconclusive; however, as far as I could make out with such data as I had, this practice neither increased the tendency to stammer nor diminished it.

## CONCLUSIONS.

*1.* By having him repeat four times in succession a panel of 25 selected test words, accurate percentage data can be obtained from any stammerer with reference to his speech errors.

*2.* On recording separately the speech errors made on four panels, successively, it was found that the values tended to pinch out in the direction from left to right.

*3.* If a stammerer repeated once daily, four times in succession, a panel of 25 test words, suddenly — usually in the course of a week — he became able to pronounce them without a single error.

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## SOME RECENT IDEAS ON DEAFNESS.

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The development of the field of otology has been strangely one-sided. Pioneers in this work had to do chiefly with the removal of wax and the local treatment of discharging ears. Surgical otology came as a concomitant of general surgery — in fact, the first mastoid operations were performed by general surgeons, and antedating that was the famous Wilde's incision for subperiosteal abscess. When it was discovered how excellent are the results from thorough evacuation of an empyema of the mastoid cells, monographs and books, notably those of Schwartze and Stacke, began to appear in considerable quantity, and men with good surgical training found this an interesting and profitable field for the exercising of diagnostic acumen and surgical skill.

Early important contributions to the subject were made by Adam Politzer; first, by the then remarkable "Wandtafeln" or wall plates,* published in 1873, setting forth the anatomy and pathology of middle ear suppuration, and later by his comprehensive book on otology, which to this day remains a classic in the particular, essentially limited, aspect of the subject therein described.

So-called "medical otology," save for treatment of acute suppuration in the middle ear, has remained from the first a difficult and recalcitrant subject. Present-day treatment of deafness does not differ much from that used by our grandfathers. It is common knowledge among aurists that our hand is on the doorknob as soon as we have made a diagnosis of *otosclerosis*, or, if hardness of hearing be known by another name and is accompanied by tinnitus aurium, we hope to be spared the painful experience of trying to evade the questions of our importunate and disturbed patient.

The hopelessness of much "ear trouble" is a spectre from which we are always trying unsuccessfully to escape. We

*When in Vienna in 1910-1911, I purchased what was said to be the last available set of these plates. They are stained with age and tender of fibre, and yet are as worthy of careful study as any black and white drawings or sketches which have been brought forth in later years.

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ought to admit our shortcomings and aim to overcome them; ought to have done so long ago, but we have inherited an attitude toward deafness from the great teachers abroad, and have been content to delve into such obstruse subjects as the movement of the peri- and endolymph, and the function of the hair cells in the organ of Corti — not for their practical value, but for their academic interest. In Vienna, excellent courses were and are still given by many great teachers. They encompass the static labyrinth, the function of the cerebellum, the origin of the quick and the slow component in nystagmus and, of course, the surgery of the major complications of suppurative labyrinthitis and brain abscess. Microscopic anatomy was and is intensively studied there, and gross pathology of the ear is entered into with enthusiastic detail in the mortuary; but, as Prof. Heinrich Neumann once remarked. "We must not forget that the ear also has an auditory function!"

Diagnosis in Vienna and in all great teaching centres has been reduced to a science in itself, especially functional diagnosis of the static labyrinth. But as for diagnosis of middle ear conditions and their treatment, we have scarcely advanced beyond the days of Adam Politzer.

What we wish to find out from hearing tests is whether such impaired hearing as exists in a given individual is of middle or inner ear origin; for it is, or has been, generally conceded that inner ear deafness means degeneration of the auditory nerve (chronic progressive deafness), and that middle ear deafness is nerve-blocking rather than nerve-degenerative in nature. Were it not for diagnosis, there would be no point in doing these tests at all. When diagnosis has been established, we may then better determine whether we can help the patient by treatment. There are, of course, mixed types which combine middle and inner ear deafness; in fact, from a practical standpoint all cases of advanced deafness are "mixed." Middle ear deafness if not of an acute infectious nature is often, but not always, due to pathological changes in the mucous membrane of the tympanum. In such cases there is a marked degree of secretory change, dependent upon an overactivity or underactivity of the mucous glands in the tympanic cavity. Limited motion or fixation of the ossicles is a change secondary to the original inflammatory process. Thus a *very wet* or a *very dry* middle ear is

always pathologic and, therefore, is associated with some degree of impaired hearing, greater or lesser as the case may be.

Once he has definitely diagnosed an integral ear condition, the aurist turns his attention to the question of vestibular function. It is possible, but not usual, to have loss or impairment of either vestibular or auditory function without demonstrable loss of function in the other, but as a rule both of these elements are involved in any disease process. The differentiation is more or less academic. In every involvement of the vestibular portion, certain signs and symptoms can be determined which make diagnosis quite easy. The auditory disturbance usually comes first, save in Méniére's disease.

It is rather unfortunate that we have come to think of "labyrinthitis" almost solely in terms of the *static portion.* This is probably because many such cases call for surgery — often enough *urgent* surgery — in order to conserve life or to prevent such complications as cerebral or cerebellar abscess. We do a mastoid operation, or should do, it in order to conserve hearing or life, but in doing a labyrinth operation we cannot expect to accomplish much for the conservation of auditory function. Very often total deafness is already present, and if any portion of the hearing returns we are inclined to bless our stars, rather than our efforts, for this benign result.

Surgery of the petrous bone has made enormous strides in the past quarter century, but chiefly, I think, as a life-saving measure; for, after a necessarily extensive surgical attack, not much of the original function can be expected to remain. Just as removal of an eye may save a life, or the sight of the other eye, so removal of a labyrinth, or more correctly its "drainage," saves life or removes the complications which endanger life.

All one can do here is to take a bird's-eye view of the aural field, so to speak, and analyze the situation of both patient and aurist as it now exists. The subject is extensive and complicated, contains many angles into which we cannot penetrate, and is a sort of proving ground for those who are fond of argument. Even among advanced students of otology in Europe, there used to be wide and sometimes bitter controversy over the question whether every case of suppurative

labyrinthitis ought to be operated upon, or whether one should do it at all, since the "deaf and dumb asylums" were always populated by patients who had recovered, or at least had lived, after, let us say, a meningitis complicated by invasion of the inner ears. Twenty-five years ago, few if any surgeons would have operated upon a patient with meningitis. But recently some hope has been offered through spinal and foramen magnum drainage, and, undoubtedly, these measures do save a life now and then.

But to get back to the problem of the deafened, it must be generally conceded that our tests have been and still are not entirely satisfactory. For practical purposes, the use of the whisper and conversation voice in a quiet room give us the best clue to the practical status of a patient in relation to his fellows. All of the tuning fork tests are subject to wide variations, and the personal equation, both of the tester and the tested, are to be weighed in the balance.

For example, the *Weber* test is often hard to elicit, for the patient thinks he should lateralize to his better ear, or, if the ears are seemingly equal, "Weber is heard in the head."

*Rinné* is very often negative, but may be positive in an ear that is badly impaired; for both air and bone conduction may be shortened *pari passu*, and yet air conduction may still be greater than bone conduction.

*Schwabach* is subject to even greater variations, since the bone conduction of the examiner may and probably is not normal, especially in otologists age 50 years or older. Therefore, the examiner may set down "Schwabach lengthened" or "Schwabach shortened" and be quite wrong in his conclusions. An examiner with normal audition may find quite the reverse when checking on his older colleague.

Dr. Robert Sonnenschein, of Chicago, has made an excellent suggestion for performing the Schwabach test, which ought to be adopted by all of us. Instead of the examiner using his own bone conduction for comparison, the fork for this purpose should be "standardized." That is, it should be checked in a varied number of cases of varied age by timing the vibrations until one knows just about how long this fork should be heard when applied to a normal skull. Suppose we take an unweighted fork having, say 108 d. v. Let us assume

that at age 25 years it should be heard for 50 seconds. If it is heard for 30 seconds, or for 60 seconds, we have a measure of shortening or lengthening. The stroke which starts the vibration should, of course, be of the same force in every instance. A difference of five or 10 seconds either way is permissible as it allows for the "personal equation" of examiner and examinee. To determine this standard for a given fork, many examinations must be made to be sure, and it is, at best, subject to variations in accuracy, but is far superior to the old method.

The *Gellé* for fixation of the stapes should be one of the best, theoretically, but there are certain difficulties even with intelligent patients who seem unable to determine any change in sound between compression and release of the rubber bulb. Another difficulty, but one easily overcome, is the matter of getting a tight fit of the occluding tip in the external auditory meatus. For this I use a hard-rubber, pyramidal tip which belongs to a nasal irrigator. Upon applying a film of vaseline, it can be made to fit the meatus quite snugly. The fork is set vibrating upon the rubber (Politzer) bag. It is usually heard. Upon compression the patient is asked whether he still hears it. The conclusion is that he will still hear it if compression fails to move a fixed stapes. Under fairly normal conditions, compression gives greatly diminished or absent hearing for the moment, until release of the bulb, when hearing again returns.

*Galton's* experiment with a whistle served its purpose well before the advent of other noise-producing agents. It is now quite obsolete.

The ticking watch may give rise to blatant errors unless the distance for normal hearing for a given watch has been determined (calibrated) on a series of individuals. Few of us have access to "soundproof" testing chambers, and the patient is confused by all external sounds, and by his unfamiliarity with a "strange" timepiece.

In testing for malingering in one ear, the *Stenger* and the so-called "reading test" are both of value, since they reduce the chances for trickery to a minimum unless the subject refuses to answer any questions at all or evades cooperation.

It seems to me that no instrument has yet come to our attention which is scientifically accurate in testing via air conduc-

tion, and it is upon air conduction that most of us depend for our auditory impressions. Any instrument which must contact the ear or any other part of the body does not give air impressions only, but tactile sensation and conductivity through a solid medium (bone). This is just as true of the audiometer as of any other testing instrument. Intensity is increased by conduction over the mastoid bone as compared with conduction via the pinna and external auditory meatus. However, the audiometer represents a praiseworthy effort to obtain a more accurate estimate of hearing ability, but it will have to be improved upon still further before it obtains a perfect score.

With this little background of discussion, let us now consider the present situation of the deaf child. Indeed, we would wish to go even further back, and consider *how to prevent the deafened child from becoming deaf*. When an aurist has examined the deaf child and determined the status of all those factors which enter into the picture, he must be prepared to give proper advice to the parents; for their hopes hang upon his very word and gesture. Heretofore, we have advised lip-reading as soon as it can be arranged for, since it involves a long period of tedious training, it is seldom successfully acquired in the adult years. Visual methods of instruction include the "sign language," written words, pictures, symbols and lip-reading. Any and all of these should be combined with aural and speech training. The latter can be taught only by vocal imitation of amplified sound. Fortunately, in these latter days, since radio has made the study of acoustics imperative, apparatus is being devised which can bring a high degree of amplification to the deafened ear. Then the imitative instinct comes into play, and the child tries to reproduce the impressions he has obtained. It is at once obvious that anyone who undertakes the education of the very deaf should have excellent speech, especially as to clarity of syllabic enunciation. A course in phonetics is indispensable to the teacher who intends to make education of the deaf a life-work.

One thing is certain — namely, we cannot hope to obtain satisfactory hearing by any given method unless we stress the importance of speech. Speech and hearing are complemental. Satisfactory hearing brings about the urge to reproduce what has been heard, or at least to rephrase the ideas in one's own language. The effort to speak is a great aid to

the effort to hear. No one who has "listened in" on a hearing lesson can ever forget the delighted expression on the faces of the handicapped when they perceive their own voices vibrating in response to an act of the will. It is one of the factors which make this sort of teaching satisfactory beyond compare, and is sufficient reward for any sacrifice that a teacher may make in time and patient effort.

Some educators do not approve of auricular training for reasons which are satisfactory to them: *1.* The manual method has stood the test of time, and is quite good enough for the small circle of deafened who can make use of it. *2.* Auricular training is prohibitive because it is costly, both in time and money. *3.* For many years the "sign language" in the presence of good vision has been considered adequate for any kind of teaching, from ordinary school to the higher academic curriculum leading to a degree. *4.* Heretofore, amplification of sound by means of electrical apparatus has been unsatisfactory because of distortion and certain other limitations inherent in the ordinary amplifier, microphone and receiver. Moreover, the instructor has been unable to estimate and to control the amount of amplification exactly suited to his pupil. And in class work such control has been well-nigh impossible. Therefore, neither educator nor otologist has recommended auricular training save in a few instances where persistent effort has shown that it is both possible and preferable.

An otologist who has translated the courage of conviction into action is Dr. Max A. Goldstein, of St. Louis. In his well known Central Institute for the Deaf he has made use of the ordinary stethoscope, fitted with a mouthpiece for the teacher and aural receptors for the pupil. Sound amplification has been obtained by the Goldstein method through vocal efforts of the teacher, and has been quite satisfactory, but with great expenditure of energy. Anyone who has spoken in a large auditorium at top voice for an hour knows the fatigue which ensues, and this is greatly increased if the speaker has little or no knowledge of voice production. Modern engineers seem capable of performing all kinds of miracles, and in the electric sound amplifier they have solved the problem of converting a small or ineffectual voice into a living, vibrant, convincing means of communication from man to man. Among vocal educators one commonly hears the saying, "The radio has become a godsend to voiceless singers."

This means that "small voice" can be stepped up to a booming, intensive explosion which is capable of making the listener dizzy if it be amplified to a high degree.

As otologists we have not yet "sold" ourselves to teachers of the deaf. As a class they see no advantage in our examinations or treatment. To them a discharging ear may be an inconvenience, but it is "just one of those things" which happen to people. As for impacted cerumen, it is unimportant so long as one can make his voice carry to the dormant auditory centres of the taught. In other words, diagnosis counts for little, but teaching methods are paramount. This is an error which we are in duty bound to correct. Every deafened child, no matter what the degree of impairment, should undergo a thorough examination of the ears, nose and throat, followed by correction of all physical defects which can be remedied either through medical or surgical treatment. Naturally, under such competent study, one will find not only local conditions such as sinusitis, diseased tonsils and adenoids, and malformations such as cleft palate, but endocrine disorders and other conditions which belong in the category of general medicine.

But, assuming that a thorough examination discloses nothing abnormal, let us say that both membrana tympani are normal, that no pathologic process exists in the nose or throat, and that a complete physical examination is quite negative. what can the aurist do? Well, he must use the spoken word with different intensities at varying distances, or experiment with sounds produced by an audiometer. However, conversation voice being at certain levels for all normal hearing ears, is, in the last analysis, "the one thing needful." What we actually require is a sound meter which will tell us what intensity is imperative in order to produce an impression on the brain of a given subject at the average level of ordinary room conversation. This is now being perfected by acoustic engineers and may soon be a part of our armamentarium. It will reveal on paper the intensity of speech sounds in *decibels rather than in percentages*. Now, the decibel is the unit of intensity of sound which gives numerical value for variations of loudness of the speaking voice. These variations are: whisper, soft sound, moderately loud sounds and very loud sounds. They may also be termed light, heavy and severe. In music they would be known as pianissimo, piano, mezzo-

forte and fortissimo. One should know music to know acoustics and its relation to normal speech and hearing.

The value of studying any subject academically is a worthy object of intellectual endeavor, but what present-day education needs above all else is an integration and correlation of the sciences as they now exist. If we could bring this about, unification of what has been already learned in the various sciences would produce a changed world, and greatly increase the element of human happiness. Unfortunately, so much of so-called "research" ends in futility. Facts are determined with great precision, and then are stowed away on a dusty shelf without being brought into correlation with allied facts. Our knowledge has become so voluminous and so highly specialized that we are in some danger of being engulfed by it, and thus we may defeat the very ends for which so much arduous labor has been expended. For example, chemistry is nearly as old as civilization, but only within the past quarter century has it been made more effective in serving the health needs of man. Mathematics has always been more or less of an abstraction for college undergraduates, save those who have taken up engineering or construction. But Einstein and others have shown that mathematics is a vital element in understanding the universe, and that it has many applications in daily life beyond the realm of "pure science."

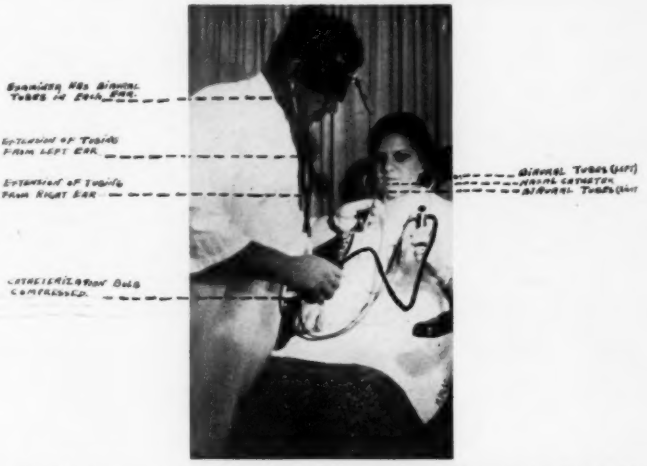
During 1919, in a town in Jugoslavia called Agram, 120 patients residing in an institute for deaf mutes were tested by radio receivers. Of these, only four did not hear any vibrations which could be interpreted as sound. But, of course, that was 16 years ago, and electrical apparatus for scientific experimentation was existent then only in a relatively crude form. It would be interesting to repeat the experiment under present-day conditions. This problem is being quietly solved by laboratory investigators. Already, deaf mutes can be made to hear and speak through electrical amplification of sound, but practical class room application is not, as yet, at hand because of many unwieldy factors which must be disposed of. What Dr. Max A. Goldstein has done by way of the stethoscope and shouting will be more effectively carried out bv electrical amplification eventually. This seems like a hasty, hazy prediction, but it is based upon well grounded facts.

140 East 54th Street.

## A DOUBLE BINAURAL DEVICE AS AN AID IN CATHETERIZATION OF EUSTACHIAN TUBES.

DR. M. MARTYN KAFKA, Brooklyn.

Over a period of several years, I have been greatly handicapped by the use of the old type of catheterization tube for the external auditory canal used for patient and the examiner. On many occasions during the catheterization I have noticed particularly that either the part fitting the patient's external


Examiner has Binaural Tubes in Each Ear
Extension of Tubing From Left Ear
Extension of Tubing From Right Ear
Binaural Tubes (Left)
Nasal Catheter
Binaural Tubes (Right)
Catheterization Bulb Compressed

A double binaural device as an aid in catheterization of the Eustachian tubes.

auditory canal or the part fitting the otologist's ear is not only inconvenient during the treatment, but that the patient occasionally feels the ear piece loosening and attempts to hold it in place. By doing so, he crosses over the opposite hand to the side of the tube and often interferes with the process of catheterization. This interference may cause injury to the patient's orifice of the Eustachian tube by forcing the metal

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catheter into the nose. By the use of this double binaural device the patient has two hands free, and so can aid the examiner by holding the Politzer bulb, or anything else that may be necessary during the catheterization process.

The patient feels more at ease by having the device in each ear simultaneously. There is also the added convenience to the examiner, who at the same time has binaural tubes in his ears. The rubber tube can be made very long. The ear piece fits snugly and can be made compressible without interference of body or the head motions.

I have found this device a great aid in my work, especially so in various types of conditions where snugness of the ear piece is essential in the air reflexes during the catheterization.

807 St. Marks Avenue.

# IN MEMORIAM

## ALVIN J. LORIE, M.D.

1886-1937.

Dr. Alvin J. Lorie, of Kansas City, died at Miami Beach, Fla., March 9, from a heart attack.

A native Kansas Cityan and a graduate of Westport High School, 1904, he attended the University of Michigan and was an instructor in oto-laryngology there for four years following his graduation. His excellent training in these specialties was developed under the preceptorship of the late Dr. R. Bishop Canfield. In 1912 he began his oto-laryngologic practice in Kansas City. On April 15, 1917, he was married to Miss Adele Hirsch, of Denison, Tex.

Dr. Lorie was vice-president of the Midwest Section of the American Laryngological, Rhinological and Otological Society; president of the Kansas City Society of Ophthalmology, Otology, Rhinology and Laryngology; a director of the Ear, Nose and Throat Department of the General Hospital of Kansas City for the past 20 years; chief of staff of Menorah Hospital until his resignation a year ago; a Fellow of the American College of Surgeons and a member of the American Academy of Ophthalmology and Oto-Laryngology. He was a past president of Phi Rho Sigma and Phi Kappa Sigma, medical fraternities; a life trustee of the United Jewish Charities and a member of the Jackson County and Southwest Clinical Societies.

Last November he was elected Coronor of Kansas City, the culmination of many years' interest in the medico-legal aspects of his home city.

It was a privilege to know this capable, genial man and humanitarian, a forceful influence in the scientific and charitable activities with which he was identified; a lover of the out-of-doors and an enthusiastic sportsman; a keen reader of human nature; a highly trained scientist and a loyal friend.

He was a man of modest demeanor, fearless in the expression of his convictions and possessed of unusual executive ability.

He was a founder of the Alfred Benjamin Dispensary and Menorah Hospital and continued his active interests in these philanthropies to the end.

We knew him as a devoted husband and father, as a highly respected and unusually capable colleague, as a medical idealist and champion of high standards and as a devoted friend.

These memories will perpetuate themselves not only in the hearts of those of his intimate circle and in the community in which he has made such an enviable record, but also in the entire otolaryngologic world, where his substantial counsel, wide clinical experience and careful scientific training made him much beloved. M. A. G.

## THE NEW YORK ACADEMY OF MEDICINE.

*Meeting of Jan. 20, 1937.*

SECTION OF OTO-LARYNGOLOGY.

**Hearing and Its Conservation in the Schools.** Wm. E. Grady.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

**National Organizations for Service to the Hard-of-Hearing.** Betty C. Wright.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

**Local Organizations for Service to the Hard-of-Hearing.** Annetta W. Peck.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

**Service in the New York City School for the Deaf.** Dr. Jay D. Whitham.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

**Fundamentals of Lip-Reading.** Estelle E. Samuelson.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

**Parent and Teacher Training.** Irene B. Young.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

**The Role of the Prevention Clinic.** Dr. Westley M. Hunt.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

**Rehabilitation of the Deaf Child.** Dr. Max A. Goldstein.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. ROSS FAULKNER: I do want to say, as representing the audience, that we are very grateful for this program. It is one of the most interesting programs which I have ever heard given. I may be prejudiced because of my own infirmity, but I think that more good can be done with this than with almost any other sort of program. I think the work these people are doing is the greatest in the world and, economically, the most profitable. I fear that we, as specialists, can take little credit to ourselves in this field. Our own work lies more in the prevention of deafness. I feel that with us the best chance of accomplishing much is to get people who are young. The older ones offer rather less promise. Probably we should not ever despair, for even if we cannot help the physical defect in hard-of-hearing patients, we should bolster up their morale and send them to some of these fine social agencies which do such a splendid work in the rehabilitation of these people. This is our part as citizens, and our part as specialists is to further in every way

possible the prevention of deafness, in private practice as well as in clinics; to catch them young, treat them as gently as possible, teach them lip-reading, and give them every possible medical aid we can.

DR. HUGH G. ROWELL: On an evening of such significance as this in the work with the deaf it is rather difficult to add anything to what has already been said; however, I have two announcements which will perhaps be of interest on this occasion, and are a tribute to the splendid work which is being done. Late in June we shall act as hosts to the convention of the American Instructors to the Deaf, and in July or August we shall also be hosts to the annual summer session of the American Association to Promote the Teaching of Speech to the Deaf.

In the Department of Education of the Handicapped at Teachers' College, Columbia University, we feel first of all that the proper background for any teacher of the handicapped is that of the teacher of a normal child. This may seem, at first, to stretch the point pretty far in the case of the teaching of lip-reading; however, the responsibilities of any teacher of the handicapped are even broader than those of the regular teacher, and we can no longer accept in our schools the individual whose professional preparation for the instruction of the hard-of-hearing is a brief elementary course in lip-reading methods. In our own curriculum we require a thorough appreciative knowledge of the ear and vocal mechanisms from anatomical, physiological and clinical points of view; we want them to know the ordinary tests of hearing used in schools, and to understand something of the interpretation the aurist places on these; we want them to serve as guidance experts, both educational in the broad sense, and perhaps vocational as well. We want this teacher to know something of methods of instructing the deaf. It is a big job.

We have not, as yet, attempted to train teachers for the nursery schools or adult education projects in the education of either the deaf or the hard-of-hearing. It will be necessary to do considerable experimentation before we would be willing to set up a criterion as to just what we want. We do know this — a good deal of specialized background will be necessary, and also a thorough grounding in general education; however, we fully appreciate the importance of this work. When this Department starts training personnel for those areas, it will work in cooperation with our kindergarten-nursery school and adult education departments. Also, as is our custom, we shall endeavor to work closely with those persons who may be considered leaders and authorities in the various phases of the training program.

I welcome this meeting tonight as a generous gesture on the part of the Otological Section of the New York Academy of Medicine to give full recognition to the importance of educational programs in the otological picture. The educator is always better satisfied when he is working in cooperation with the medical profession, and this is more true in connection with the education of the handicapped than in any other field. We, who have been in the conservation of hearing movement for years, feel gratified at each new evidence that our efforts have not been wasted. We feel in our department at Teachers' College that the otologist more and more will recognize definitely the therapeutic value of properly planned and administered educational programs for his deaf-blind, deaf, or hard-of-hearing patients. On our part, we propose that we shall seek only the finest and best trained for this new adjunct to medicine.

DR. JOHN W. DURKEE: It seems to me that the question that might very well be asked at the present time is "Why another clinic?" Why not treat these patients in the regular ear, nose and throat clinic? In an active ear, nose and throat clinic, where two to three hundred patients are being seen in an afternoon, most of the attention must be centered in those who are acutely ill. If anything is neglected or pushed aside, it is the chronic case of hard-of-hearing. I think there is room for a hard-of-hearing clinic to which these patients can be referred and treated carefully. Dr. Hunt, in mentioning the requirements for the clinic and the things that should be in it, did not emphasize the thing that I think most important — the man who runs it. He must

be a physician who is willing to take time, make a thorough examination, and talk with the patient or with the mother. These patients who come to the clinic or to the physician as a rule expect to be cured; sometimes you have to tell them that they cannot be cured, and then you have to treat not only the ears, but the individual. We have to explain to these patients, or to the mothers, what is ahead of them, what can be done, and then help them to adjust their lives.

DR. AUGUSTUS J. HAMBROOK: The Deaf and Hard-of-Hearing Sub-Committee of the Medical Society of the State of New York, a committee made up of members of both the Health and Public Relations Committees, was formed about three years ago. Its first chairman was the late lamented Dr. Wendell C. Phillips, a pioneer in this field. The main objectives were for raising the standard of otological care of children in schools for the deaf and for the conservation of hearing of all school children in the state. A survey of the schools for the deaf was made and it was found that many children were admitted without an otological examination, and very little care was given children with ear conditions requiring treatment. Many children in the deaf schools were not deaf but hard-of-hearing. Their education in such schools was to their serious disadvantage as well as to that of the deaf children. Careful otological examination before admission and special treatment of any diseased conditions of the ears will correct the above.

The question of hard-of-hearing children in this state, we feel, is a real problem. We have about 3,500,000 children in the schools of the state, and from accurate surveys made in various parts, from 5 to 10 per cent were found to have some degree of hearing impairment. We are concerned with children whose hearing is threatened (potential impairment) and those who have lost a certain degree of hearing (an acquired loss). To make possible the early discovery of all children deafened or hard-of-hearing, legislative measures were passed last year and signed by the governor. The two bills passed provided: First, for annual examinations of hearing acuity of all school children, scientific methods such as the audiometer to be used; second, requiring the reporting of all deaf or hard-of-hearing children to the Health Department. A third bill failed of passage. This would have provided for the early admission of all deaf children to a deaf school. Thus proper education would be started at an early age, to the benefit of the deaf child and the state as well. Provisions were included for lip-reading instruction in the regular schools, for moderately hard-of-hearing children, and special classes in the regular schools for children with too great a hearing loss to profit properly by grade instruction unless aided by lip-reading and hearing equipment. We hope to have such a bill passed this year and solicit your active help and cooperation when the bill comes up in the legislature at Albany. The last House of Delegates of the Medical Society of the State of New York voted to petition the governor to appoint a commission to survey and supervise the care of deaf and hard-of-hearing children in the state. The governor felt that he was completing his services to the state and took no action. We hope to have such a commission appointed this year.

DANIEL CAPLIN: The work of the WPA Project 6065, Conservation of Hearing of School Children, of which I have been the technical director since January, 1934, has proven to practically all connected with it, directly or indirectly, the need for testing the hearing of our school children, and for treatment of pathological conditions of the ear, nose and throat in order to conserve hearing; however, I am taking this opportunity to ask you, the medical profession, to give more earnest and favorable cooperation, even though in a great number of instances such cooperation has been most freely given and, I assure you, greatly appreciated by me.

The 1,000,000 or more 4A audiometer tests followed by the 30,000 2A tests and 26,000 ear, nose and throat examinations of our school children above the 2B grade have proven that much can be done for the conservation of hearing if we educators and you physicians work in closer harmony. My 27 years' work in school health education and our conclusions based on the histories in the

cases with hearing losses detected by our several tests and otological examinations show that these hearing losses were due to causes that started in the pre-school period. It would be presumptuous on my part to say that in the majority of instances the so-called children's diseases with complications are by far the most frequent causes of the hearing loss, but that is what you otologists have taught me. From the 30,000 cases examined with the 2A audiometer, I have chosen at random one of our largest schools for detailed analysis, and will give you the data on which I base the above conclusions. In this school, 2,000 children were given 4A audiometer tests; 232 children were given 2A audiometer tests, followed by otological examinations. The past histories of these 232 were as follows:

| Age | Measles, Scarlet Fever, etc. | Pain in Ear, Discharge, or Both |
|---|---|---|
| 1 | 45 | 12 |
| 2 | 35 | 12 |
| 3 | 32 | 5 |
| 4 | 38 | 9 |
| 5 | 22 | 3 |
| 6 | 29 | 8 |
| | 201 | 49 |

For all years, only 31 had negative histories. The majority of the above 201 children had either measles, scarlet fever, or one of the other causative illnesses later, but I have taken the earliest age at which one of the illnesses occurred in order, if possible, to correlate it with the onset of the ear trouble.

The hearing loss in these children at the time of the 2A audiometer tests was as follows: Five to 20 decibels, 135; 21 to 30 decibels, 71; 31 to 40 decibels, 17; 41 or more decibels, nine.

A glance at the above should, in my opinion, convince the most skeptical of the fact that most hearing losses started during the pre-school period, and that their detection by the available tests and, most important of all, the diagnosis and medical care of these children by you otologists, would go a long way towards solving this problem. At the same time it would save the school authorities large sums of money that they must now spend educating these children as handicapped children.

Dr. Emily A. Pratt: I was very grateful to Dr. Hambrook for bringing to your attention that in New York State we have a law for the annual testing of the hearing of school children.

This state at the present time is taking the lead, through legislation, for the deaf and hard-of-hearing child. Since 1913, in New York State, it has been compulsory that all public school children's hearing be tested once during the school year. In 1924 the position of "Eye and Ear Specialist" to the State Education Department was created—the only state in the Union with such a position, even to the present time. In 1936, the Education Law was amended so that these annual tests should be applied not only to public school children but to all children, and that the tests should be made by audiometers. We have a long way to go, however, and it will take time to administer this law and to educate towards all that it implies. We must not stop with audiometer tests but must carry on until these tests are followed by good otological examinations with itinerantly taught lip-reading for those who need it.

I appreciate Dr. Durkee's remarks pointing so definitely to the need which faces us. You people here in New York City—you otologists especially—do not realize how many more advantages you have than the upstate otologist—clinical facilities and contacts that the upstate otologists do not have. Preventive facilities through well organized clinics should be available to all pre-

school and school children. Now that we have compulsory audiometer tests for our school children, these tests are being made more and more each year. To give you a concrete example, the school registration of a certain city is 5,500. Sixty-four children were examined by an otologist after 4A and 6A audiometer tests. Those 64 children can be divided into groups showing their need: Thirty-five children with a hearing loss of 20 to 35 per cent in the better ear; 11 with a hearing loss of more than 35 per cent but not more than 70 per cent, none so deaf as to need to be placed in a special school; 11 with a hearing loss of 15 to 20 per cent, and seven with less than 15 per cent, in five of whom a poor prognosis had been made. Disregarding the borderline cases, there are 46 children in one upstate city who should receive lip-reading and for whom any possible preventive treatment should be available. It so happens that these children will be well taken care of, either privately or by public welfare. Even the borderline cases have just as much need for treatment, but it is easier to show need for outstanding cases.

We depend upon such groups as those represented here tonight for the guidance in legislation which will provide for the pre-school and school child.

Dr. Frederick N. Sperry: The care and training of the deaf and hard-of-hearing child is a most interesting and timely subject.

As an officer in the American Society for the Hard-of-Hearing, I am particularly interested, for the organizations for the hard-of-hearing have brought to the front this great public movement. Since Leagues for the Hard-of-Hearing have been formed, we have become lip-reading conscious and hearing-aid conscious. They have been instrumental in bringing out audiometers and have watched the growth of practice of testing the hearing of school children. These things have happened because the hard-of-hearing have organized and have expressed the need for social, economic and educational betterment. They are doing a grand work.

It must be apparent that otologists could not inaugurate such welfare works as we have heard about tonight, but they can be of very great help.

Mention has been made of the testing of the hearing of pre-school children and it has been said that no good test exists. This is not wholly true. There is on sale in New York at Meyrowitz's, and in Chicago, at Mueller's, a stop-watch that has been standardized and with which we get results which check quite accurately with the group audiometer. It has the advantages of easy use for the pre-school child and the price is only $4.

I want to commend what Dr. Durkee has said relative to the otological examinations. They should be made by one man who has the help of the school nurse with the audiometer records. The school nurse then can conceive the recommendations and see that they are carried out.

Something has been said about legislation. It is no secret that this government has become social-minded. There will be introduced into the present Congress a Bill for the aid to the physically handicapped. In its present form it calls for annual appropriations of $11,580,000. This Bill would call for appropriations from State Legislatures and in all of this work our Chapters will be influential.

I would be a very poor officer of the National Society if I did not take this opportunity to say that there is one way in which we, as otologists, can cooperate. We should not only be stimulated by a symposium such as this, but should have literature on our waiting-room tables—something like the pamphlet put out by the American Society for the Hard-of-Hearing. A membership which costs $1 annually will bring to you "Hearing News" every month. Regular membership costs $2. The American Society for the Hard-of-Hearing cordially invites all to join us in this crusade for the betterment of the hard-of-hearing.

## THE NEW YORK ACADEMY OF MEDICINE.

*Meeting of Feb. 17, 1937.*

SECTION OF OTO-LARYNGOLOGY.

**The Clinical Picture of Diseases of the Labyrinth Wall.** Dr. Edmund P. Fowler.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

**Disease of the Labyrinthine Capsule; Pathological Changes.** Prof. F. R. Nager.

*(To be published in a subsequent issue of* THE LARYNGOSCOPE.*)*

DISCUSSION.

PROF. FELIX R. NAGER: It is quite true that these microscopic researches have not given further knowledge of treatment, but I hope we are on the way and, from the clinical standpoint, our researches had certain interests, insofar as we saw that otosclerosis may be seen in "mute" regions, wherefrom the hearing is not affected. Moreover, we could prove that otosclerosis is not necessarily an affection of both sides, because in a certain number of cases the microscopic investigation showed the disease only in one ear.

In the demonstration we have not spoken of experimental changes of the labyrinthine capsule; however, it is more and more known that bone alterations may be produced experimentally and even the labyrinthine capsule will be affected. Now, if we can change the bone, it might be possible to cure the changes again.

The early diagnosis of otosclerosis is, of course, of the greatest importance, but in many cases where the process does not affect the stapedovestibular joint, the patient will not feel anything from the disease. The special observation of otosclerosis in mute regions has a great importance as to the laws of heredity, drawn from histories of deaf patients. I think we should be very careful in our conclusions, especially as to preventive problems like forbidding marriage, etc.

The remarks of Dr. Gutman about the biochemistry of the bone diseases are very interesting, and I hope that perhaps by other examinations we may find some similar alterations in otosclerosis.

The slides of Dr. Fowler were most interesting to me. His method of investigation seems to be very important for the studies of bone structure.

Dr. Dwyer spoke of the affection of circulation, which seems to me undoubtedly of greatest importance. I recall the late Dr. Albert Gray, of London, well known British otologist, whose idea was that vasomotoric troubles were the cause of otosclerosis.

Dr. Kopetzky's remarks on prenatal influences or constitutional factors are very important. It is hoped that further studies may give a better light on this subject.

As to the frequency of otosclerotic foci, I remember two results of our own researches. In a number of patients who died from fractures of the skull we found three per cent with otosclerotic foci, and, during a study of patients of

all ages who died by drowning, we found even a higher percentage; so that I came to the conclusion that otosclerosis, at least in our country, is not so infrequent as it was supposed.

As to Dr. Kopetzky's remarks about atavistic signs in otosclerosis, there is the objection that in animals no signs of this condition could be found. All the similar diseases in animals described as otosclerosis were later on found as normal or inflammatory bone affections.

DR. STACY R. GUILD: Certainly by now you are aware, even if you did not have the pleasure of knowing him previously, that there is no one better qualified, or I might say as well qualified as Prof. Nager, to speak upon this topic. The wealth of his material on diseases of the otic capsule surpasses that of any collection of which I have knowledge. And his studies of this material are known the world over.

The discussion which I had prepared is completely out of place because Prof. Nager did not speak of certain things which I had expected he would. One thing which I had expected Prof. Nager to emphasize particularly I wish to bring up an addition to his presentation, especially since it is his own work. I presume this phase of the topic was omitted only because it is very difficult to demonstrate the facts with lantern slides, or indeed with anything but the microscope itself. In a series of publications by Prof. Nager, jointly with Dr. Max Meyer, of Würzburg, in 1930, 1931 and 1932, the general topic of diseases of bone and the relation of otosclerosis to the systemic diseases of bone was developed in extenso. They utilized not only hematoxylin-and-eosin stained material, such as Prof. Nager showed tonight by means of his beautiful color process photomicrographs, but also the most recent techniques for the study of normal bone and of pathologic bone in the rest of the body. The point is made repeatedly that in such a study one must observe the fibrillar structure, i. e., study the bone from the viewpoint of its finest structural elements. The facts given clearly link otosclerosis with von Recklinghausen's disease and with Paget's disease. The conclusion is irresistible that structurally the lesions of the three diseases are the same, except with respect to extensiveness and location.

As Prof. Nager has said, for the otologist otosclerosis is the starting point in these studies and also the condition of primary interest. Clinically, and also from the laboratory standpoint, the other conditions he has discussed tonight are rarities as compared to otosclerosis. Though the complete argument was not presented tonight, Prof. Nager has shown that otosclerosis is a variety, or suborder, of ostitis fibrosa generalisata (von Recklinghausen's disease of bone). They are the same in essential morphology and in the history of one transformation after another until the end, or quiescent, stage is formed. The very fact that the two diseases are morphologically similar makes possible the utilization by the otologist of anything basic that is learned about the more widespread systemic condition. But, as Prof. Nager himself said in the beginning of his presentation tonight, with all our knowledge of structural sequences and morphology we know nothing of the cause of otosclerosis. I read from his work in a free translation, as follows: "Of the cause we know even so little as of the disease itself." Neither do we know the cause of von Recklinghausen's or of Paget's disease, both of which, by the way, have some tendency to be hereditary, though not to the degree that otosclerosis is.

It is always the hope of medical men, and at the same time the ultimate purpose of all laboratory investigations in medical fields, that the new discoveries or factual observations with respect to morphology, biochemistry, etc. will lead to a more rational basis for intelligent treatment and/or prevention of disease. From the standpoint of treatment or of prevention of otosclerosis we are in the same position as we were before. It is Prof. Nager's hope that his work will stimulate further investigations rather than that this phase of the topic will be called finished. His own studies raise new questions, as does all good work.

Of special interest from the standpoint of treatment or of prevention is Prof. Nager's statement that only 45 per cent of cases with histologically proven otosclerosis show an ankylosis of the footplate of the stapes. My own experience in the laboratory indicates an even smaller percentage, perhaps 35 per cent. It has been generally recognized for 20 years or more that unless there is ankylosis of the footplate of the stapes there is not the characteristic impairment of hearing, if indeed there is an impairment at all. Prof. Nager has written on this phase of the topic, as have also Otto Mayer, Wittmaack, Lange and many others.

Even though one knows all about the morphology of the lesion, what is there that can be done to treat these patients, either to arrest the progress of the lesion or to restore the hearing after there is an ankylosis of the footplate? You all know the small dimensions and the location of the footplate of the stapes. Personally, I cannot conceive of any therapy, medical or surgical, which can restore to normal an ankylosed stapediovestibular articulation. From the otologists' point of view it will not be sufficient to learn how to arrest the progress of an otosclerotic area. We must also learn to recognize otosclerosis before ankylosis has occurred, which means we must learn to recognize it by some method other than tests of hearing. I leave to your imaginations what methods for this purpose may possibly prove successful. All of you, I believe, are familiar with your Chairman's studies upon the blood of patients with otosclerosis. Other studies have also appeared. If I remember correctly Dr. Fowler's conclusions of a few years ago, his patients did show variations in the calcium, phosphorus and cholesterin content of the blood but the range of these variations was the same as the variations in normal people without otosclerosis. That such studies have thus far yielded negative results is no reason for abandoning the search for a method that will permit detection of otosclerosis before the hearing becomes impaired.

As many in this audience are aware, the primary interest of the group in our laboratory at Baltimore is to correlate hearing impairment with lesions. Perhaps that is why I have tried in this discussion to stress the phase of hearing. Personally, I would be very much interested to learn from Prof. Nager more about the types of hearing impairment associated with the conditions he has described tonight, but that is really beside the point of his paper, which is on the subject of the diseases themselves. I am sure I express also the view of Prof. Nager when I say that I hope this work which he has so ably presented will stimulate men in the audience here tonight to undertake studies other than morphologic. The morphologic work is far in advance of what is known about the effect of nutritional and biochemical disturbances, etc., upon lesions of the otic capsule. I believe that the next big step in advance with respect to our knowledge of otosclerosis and other diseases of bone will be in those fields.

Again, I wish to express my appreciation for the opportunity to be here tonight.

DR. ALEXANDER B. GUTMAN (by invitation): The particular aspect of this more general subject which I should like to present is that relating to the biochemistry of the blood in the several diseases of the bones mentioned by Prof. Nager this evening.

The three points I should like to emphasize particularly are, first, what chemical determinations are relevant in connection with the differential diagnosis of bone disease? Second, what are the characteristic deviations from the normal in the several bone diseases discussed? Third, of what, if any, value are such deviations from the normal in the differential diagnosis of skeletal diseases in general and diseases of the labyrinthine capsule in particular?

Three blood determinations have been found useful in the differentiation of certain bone diseases: Serum calcium, serum inorganic phosphorus and serum phosphatase. It is often necessary to determine also the nonprotein nitrogen (or urea) content of the blood. Phosphatase is an enzyme which is

present in normal blood and, as was discovered a few years ago, in increased amounts in certain diseases of the skeleton.

We might discuss Paget's disease first because several sections showing Paget's disease involving the labyrinthine capsule were shown this evening and because of the possible implications in connection with otosclerosis. Our studies on Paget's disease include 116 cases which were treated at the Presbyterian Hospital between the years 1925 and 1935. I shall not attempt to give our clinical and X-ray findings except for one or two points of otological interest. In three of our 116 cases of Paget's disease, the initial and presenting symptom was deafness, and in 26 cases, one of the major subsequent symptoms was loss of hearing. Inasmuch as Paget's disease is not as rare a disease as had been previously supposed, Paget's disease is of some practical importance as a cause of deafness May I remind you further that there need not be any clinically obvious deformity of the cranium despite extensive involvement of the skull, with impairment of hearing, due either to compression of the auditory nerve or to destruction of bone; and that one may have extensive involvement of the skull without involvement of other bones of the skeleton. Fourteen of our cases had large heads without other bone involvement. In our experience the skull, next to the pelvis, is most frequently involved in Paget's disease.

It is a remarkable fact that although the skeleton may be very extensively involved in Paget's disease, the serum calcium remains within normal limits (between 9 and 11 mgm. per cent). Of the 61 cases in our series which we had the opportunity to examine at the time this table was made, 52 had normal calcium values. The inorganic phosphorus was within normal limits in 58 of the 61 cases, and slightly elevated in the others. The phosphatase, however, was consistently elevated. It is the only known constituent of the blood which shows any change, even in extensive Paget's disease. The Bodansky method which we use gives normal values between 1 and 4 units. In Paget's disease one obtains values as high as 50 or 100 units, the increase being an expression of the increase in bone formation found in these cases. Phosphorus and calcium metabolism studies are normal in Paget's disease. The increase in phosphatase is an important aid in diagnosing Paget's disease.

In von Recklinghausen's disease or osteitis fibrosa cystica the chemical changes in the blood are quite different. We, ourselves, have seen only seven proven cases. But of 120 cases from the literature, the serum calcium which was determined in 83 was found to be elevated in 78 instances. This is in marked contrast to the normal serum calcium values found in Paget's disease. Inorganic phosphorus was determined in 56 cases and found to be low in 28. Phosphatase determination was done in 19 cases and found to be increased in all cases with definite bone changes. The degree of elevation, however, is not as marked as in Paget's disease, being between 6 or 8 units and 40 units in most cases.

The normal calcium findings which Dr. Fowler has observed in a very large series of cases of otosclerosis would suggest, insofar as the biochemical evidence is concerned, that otosclerosis is not associated with hyperparathyroidism. I do not know whether Dr. Fowler did phosphatase determinations in his cases of otosclerosis. The determination of phosphatase might give further evidence of value, inasmuch as the sections of the capsule shown this evening did show new bone formation.

There are other bone conditions which give more or less distinctive blood chemistry values. Multiple myeloma was not mentioned this evening but may cause changes in the labyrinthine capsule, either directly or in the form of myelomatous soft tissue tumors which erode the labyrinthine capsule. A case I have in mind developed deafness while in the hospital, which we were quite unable to explain clinically. Hypercalcemia is a common but not constant finding. Serum phosphorus and phosphatase are normal. The serum protein is elevated in approximately one-half of the cases. In multiple myeloma, chemical analysis of the blood is often of value in differential diagnosis insofar as one

observes hypercalcemia and hyperproteinemia in a fairly large proportion of the cases.

In metastatic carcinoma one occasionally finds changes in the blood. We have been obliged to distinguish between two types of metastases: osteolytic and osteoplastic. In cases with extensive osteolytic metastases, and these may, of course, affect the labyrinthine capsule, there may be elevations in the serum calcium, not to be confused with the hypercalcemia of multiple myeloma or hyperparathyroidism. On the other hand, in osteoplastic metastases, which usually arise in the prostate in the male and not infrequently in carcinoma of the breast in the female, hypercalcemia is not observed, whereas moderate to extremely high increases in phosphatase are found. Here we have yet another condition in which bone formation may go on extensively and at a rapid rate and in which the phosphatase, the enzyme which has to do with bone formation, is increased.

In localized giant cell tumors, the blood chemistry is normal. This fact has been used on a number of occasions to distinguish between benign bone cysts or giant cell tumors and hyperparathyroidism. In senile osteoporosis the blood chemistry is entirely normal. In osteogenesis imperfecta the values for calcium and phosphorus are entirely normal. Phosphatase values are slightly elevated, but since we are dealing with young individuals in which phosphatase is higher normally, they are within normal limits for that age group. In osteomalacia, which may be difficult to distinguish from hyperparathyroidism, phosphorus is consistently low and phosphatase is usually only moderately elevated. Calcium is usually normal. In Schüller-Christian's disease the values are entirely normal.

In resumé, may I say then that while studies in blood chemistry in bone diseases are still being carried out, it is quite clear that the determination of serum calcium, inorganic phosphorous and phosphatase may be of value in differential diagnosis? They are of value particularly in hyperparathyroidism, Paget's disease and multiple myeloma. The determinations are useful also in excluding other bone diseases in which these constituents of the blood are within normal limits.

DR. SAMUEL J. KOPETZKY: True it is that we may not be able to sing and yet can certainly appreciate the opera. True it is that we may not be able to quote Shakespeare with the musical cadence of a good actor and yet can sit in the theater and enjoy a Shakespearean play. So I, as a clinician, never having done any of these sections, can sit here and certainly express in no limited words my appreciation of the lessons that they teach, both Prof. Nager's presentation and the natural development of it as Dr. Fowler, Jr., presented it. Dr. Nager for a generation has made it his life work, studying these pathological exhibits. Dr. Guild is provocative when he says, what are we going to do about it? Naturally, sitting as we do at the ringside, perhaps we may be pardoned for making the usual stupid comments of the observer that nothing thus far presented has shown us the living lesion. Perhaps Dr. Gutman's examinations in the blood show something which we, as clinicians, can take with us.

All these dystrophies seem to be started before we are born. They are all fetal lesions. What causes a fetus to grow normally? There is in every organism, in every germ protoplasm, a certain driving force that makes it develop. Something interferes and brings forth these monsters and dwarfs which make subsequently fine pathological specimens for study. I think most of us are agreed these are all dystrophies, and the analogy of Paget's disease with otosclerosis is so close that who dares say that they are not also of prenatal origin? That hereditary influences in otosclerosis are our prime concern we have long known, and Alexander and Gray and others who have studied these specimens, moot, suspect and surmise the prenatal origin of factors which we do not too well understand as having an influence in creating this condition. If you learn anything from the slides that Prof. Nager showed here tonight, it is that these otosclerotic lesions, unless they impinge upon the joint between the footplate of the stapes and the walls of the window, do

not give us a clinical sign. Can anyone tell us how many people, normal in every respect as regards hearing, if autopsied and the temporal bone sub ected to serial sections, might not show lesions not impinging on parts on that temporal bone enough to produce a reaction in hearing which we can estimate?

I spent two years on the otological service of the custodial institution on the Island in my salad days, and studied a good many of these dystrophies. If I had known then what I do now, what a collection of material could have been assembled! Unfortunately, these things pass through our hands; however, I noted that if one of these dystrophies succumbed locally to infection, it spread rapidly like a prairie fire and the bone succumbed. In other words, there was not that something in the bone that made it strong, virulent and germ-resistant. That is all I got out of it. We, too, played with the idea of complete skeletal X-rays on every case of progressive deafness that came into our hands for a while. However, those of you who, like myself, are not in university institutions, know what happened: The trustees soon intervened and stopped the work because of the expense. Nevertheless, we got enough specimens to prove what Dr. Gutman said, that many of those cases that come to us with progressive deafness show changes in the bony structures of the body which we cannot study further.

This symposium tonight would not be complete if one did not at least say a word about the theory of Guggenheim. I hold no brief for him, but will at least say a word for the phylogenetic theory of Guggenheim who believes that all these dystrophies and all these conditions which were so beautifully shown on these slides have a prenatal origin and are an atavism, as it were, a reversion or regression. Those not philosophically inclined are probably in the position of the ignorant tourist who stands at the edge of the Grand Canyon and listens to the erudite guide who tells him that here is the whole history of this terrestrial earth cut in cross-section for us to understand. The man who studies genes and heredity makes a statement of basic law which says that we and every other animal go through every previous stage which animals have gone through. Is there, then, no thought that perhaps the therapy resides in the control of those endocrines that govern growth? I do not pretend to know what they are. Surely we have seen the endocrinologist handle cretinism and handle the lesions produced in bone growth through the pituitary and through the thyroid. Surely the pictures that result in the bone structure from adenoma of the parathyroid are suggestive that if we are looking for a practical clinical result, the study of these endocrines at the time when they act on the new growth intrauterally might open a field.

This is an unusual symposium. We have here the master of masters, Dr. Nager, presenting his work, and there is nothing that I or anyone can say that would detract one iota from his contribution. We have the student, a man from our own group whom we admire because of his classical work, but men work by habit. Perhaps Dr. Guggenheim's remarkable little book might profitably also be studied as a contribution to the general topic of the dystrophies and their effect on hearing.

Dr. Fowler did not suggest to me that the suppurative conditions in the labyrinth would be included in the symposium. I am very happy tonight to hear Dr. Dwyer say what he did, because at one time, when Neuman was here and when the wave spread through the country on handling purulent labyrinthitis, I was the one voice against putting any patient in a category to fit a fixed formula. I think we are coming to a saner view on the topic of labyrinthitis. One picture which Dr. Nager showed tonight brings to mind another contribution of one of our own members. It was the picture in which he showed meningitis secondary to a fracture in which the labyrinth was invaded by the meningeal infection from within. Too many of us still believe that it always comes from the middle ear to the labyrinth. A good many of us know that it goes the other way, through the internal auditory meatus into the labyrinth.

EDMUND PRINCE FOWLER, JR.: It is a great pleasure to have Prof. Nager give us at first hand some of his classical researches on the labyrinthine

capsule. Anyone who has examined temporal bones through a microscope cannot help but share his enthusiasm for the subject. The labyrinthine capsule is the most fascinating bone in the body to study. In its three layers it shows practically all the variations that can be found in any bone. Even in an adult the bizarre structure of an embryonal growing long bone is still apparent. Show the average pathologist a labyrinthine capsule and he becomes more interested in the normal structure than in any pathological areas that may be present. Now this bizarre structure of the capsule makes it somewhat easier to study disease processes in the bone. The pathological areas stand out in sharp relief against the peculiar-looking normal bone so that one can be absolutely sure where the pathological processes begin and end. For example: In a case of Paget's disease even the highly sclerotic areas contain more or less well formed Haversian systems which can be easily differentiated. A small focus of otosclerosis, such as those so commonly found near the anterior border of the oval window, could easily be lost elsewhere in the body; perhaps that is why new bone resembling otosclerotic foci has never been found in other bones of the body.

It has been my good fortune to have access to the ultra-violet microscope of Dr. Boedecker in the Columbia Dental School for the study of the finer structure of bone. From this study, supplemented by a polarizing microscope and silver stains, it has been possible to make some interesting observations on bone in general and to conclude that the pathological, as well as normal bone growth, has only a few variations in its pattern and these variations are more or less dependent upon the speed of growth. Embryonal bone growth is naturally a rapid type of bone growth. It starts with a fibrous marrow which has fibroblastic, chondroblastic and osteoblastic potentialities. Depending upon the speed of growth and the stress to be applied to the bone, there develop more or less of these types of connective tissues. Typically, the fibroblasts develop a roundish shape, come very close together and lay down on one side an eosin staining, more or less homogeneous matrix. As this matrix grows it envelops the cell, leaves it connected by its star-shaped tentacles to neighboring cells. The laying down of matrix then continues and this matrix builds itself a fibrillar structure. Now in rapidly growing bones the fibroblasts often develop into large round cells, somewhat larger than the osteoblasts, and these lay around themselves a basophilic staining homogeneous substance. At first this resembles hyaline cartilage, but as the bone grows older it is either immediately destroyed and replaced by some other type of bone, or it remains for a while and takes on more and more the characteristics of a true primitive bone with an island of homogeneous basophilic substance embedded in it. These basophilic islands remain throughout life in the labyrinthine capsule and are largely responsible for the bizarre appearance of the capsule. They have been shown by Zawisch-Ossenitz, of Vienna, to be more nearly bone than cartilage; that is, the actual structure of the cells is a cartilaginous-like cell surrounded by a matrix of bone fibrils which stick out as a globular mass in a homogeneous basophilic material which resembles cartilaginous matrix. Zawisch-Ossenitz therefore recommends that this tissue be called pseudo-cartilage and that the appellation "cartilage islands" be discarded. It is most interesting that this pseudo cartilage should remain in the inner portions of the labyrinthine capsule, for diligent search throughout embryonal and adult material reveals that pseudo cartilage in other parts of the body is only present when bones are going through a period of rapid growth. It appears to be a sort of "cheap filler," which is replaced by lamellar or some other type of bone if any stress or strain is to be put upon the bone. Now the labyrinthine capsule has very little strain on it and there is plenty of periosteal lamellar bone around it to take up the average strains which occur. Perhaps this is why the "filler" remains. Perhaps this is also why no Haversian systems are present in the endochondral capsule, for, after all, the Haversian system has been shown to be a series of laminations with a structure calculated to produce strength. Now the bone that is present in the labyrinthine capsule is not in large part the most primitive type of bone; that is, short fibre web bone. It is a bone with parallel fibrils which weave in and out among the pseudo cartilage islands like so many skeins of silk. Later in life, for some obscure reason,

this endochondral capsule may break down in a few areas and when these rebuild, Haversian systems begin to appear. Pathologically, however, in deficiency diseases, in Paget's disease, otosclerosis, etc., when the labyrinthine capsule is broken down a new bone growth occurs and this may be either of a short fibre web type, that is, of very primitive embryonal type, or of the Haversian system type; and, as a matter of fact, in almost every bone disease in which the process goes on for any length of time, the process resembles the growth of a normal long bone, that is, there is breaking down of the bone, replacement by primitive short fibre web bone, which is in turn replaced by more or less well-formed Haversian systems; if the process continues the Haversian systems are in turn broken down and replaced by more short fibre web bone, more Haversian systems or by fibrous tissue. The pattern of this fundamental breaking down and repair of bone may vary, but its life cycle does not and, more important still, this is the life cycle of bone repair and pathological bone processes everywhere in the body. Occasionally, in rapidly healing fractures, there is a reappearance of the pseudo cartilage element, but the labyrinthine capsule is so small and so little subject to strain that the body rarely has to use this method for cheaply filling up areas where bone is later to be laid down.

Unfortunately, this general discussion and the specific bone diseases of the labyrinthine capsule shown by Prof. Nager do not get us to the very root of the problem. What we do not know is what makes bone break down in a specific area and then build itself up again. We do know from the research of Wittmaack and others that bone can be made to break down if the venous flow from a given area is obstructed. We do know that there is a general loss of bone with calcium and certain vitamin deficiencies. But we cannot put our fingers on the specific modus operandi in most of the disease discussed by Prof. Nager tonight. We do not know why bone destruction and bone growth, even in such destructive processes as a coalescent mastoiditis, go on side by side. When we have solved this problem we will have some idea as to the etiology of otosclerosis, osteitis deformans and the other bone diseases whose etiology now puzzles us so much.

# MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

SECTION ON OTO-LARYNGOLOGY.

*Meeting of Feb. 9, 1937.*

PROF. FELIX R. NAGER, Professor of Oto-laryngology at the University of Zurich, Switzerland, was the guest speaker of the evening and read a paper, illustrated with numerous lantern slides, on "Demonstration on Gradenigo's Syndrome."

ABSTRACT.

One of the great fears of the otologist is meningitis occurring after middle ear suppurations. In chronic ear affections the meningitis is, by our new methods of diagnosis and treatment, not so dangerous; but in acute forms we all are very fearful of this complication.

During recent years one form of meningitis has become much better known — the form which occurs when a suppuration of the apex breaks out in the subdural spaces. Microscopic examinations have shown the alterations in this form of suppuration. We know that the apex may be filled with diploetic bone tissue or with pneumatic cells, these being a part of the pneumatization of the petrous bone. From the middle ear the suppuration infects the cells, not only of the mastoid but of the apex, and if the inflammation leads to an empyema we shall have the same signs and complications as are known to occur in the mastoid cells. The microscopic alterations are very distinct. The outbreak may occur in the middle fossa where the ganglion gasseri lies, or in the posterior fossa via the internal meatus. Both complications will lead to meningitis. Our aim should be to recognize this form of suppuration before it spreads to dangerous regions or assumes dangerous proportions.

Nowadays we have the clinical symptoms due to the retention of the suppuration. Moreover, we might find Gasserian symptoms and, as it is known in Gradenigo's disease, a palsy of the VIth nerve.

The X-ray examination may show distinct progressive alterations, especially if they are repeated, and it should be strongly emphasized that a distinct destruction of the superior borderline of the bone means danger. Therefore, if possible, we should recognize this state to prevent a fatal complication.

As to the treatment of this complication, the demonstrated microscopic slides show the principle of approach. If these cells are located more toward the posterior fossa, we shall have to follow this region. But if, on the other hand, Gasserian symptoms or even a distinct palsy of the VIth nerve are present, of all the different ways of approach I should recommend the method of Ramadier (1932, Society of Otology in Paris) which consists of exposing the internal carotid and, after pushing the artery to the side, entering in the apex. If after this operation a silver wire is placed in the apex, the X-ray examination will show exactly the extension of the abscess. (Demonstration of a number of X-rays.)

DISCUSSION.

DR. ARNOLD SCHWYZER (by invitation) asked Prof. Nager if the bacteriologic quality of the infection makes any difference.

PROF. NAGER replied that in all their cases they found the streptococcus hemolyticus, which corresponds to the findings of other authors.

DR. J. F. CURTIN asked Prof. Nager how much abducens paralysis he has seen with the so-called red dura secondary to a zygomatic involvement. Has he seen any of these cases of abducens paralysis without an inflammation of the pyramid?

PROF. NAGER said he has not seen cases of palsy of the VIth nerve following ear suppuration without inflammation. In childhood, Gradenigo's syndrome, even with palsy of the VIth nerve, seems not to be so dangerous as in adults, because in childhood this disease may heal after a simple mastoid operation; so the exposure of the apex may be reserved for advanced cases of adults as above described.

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